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THE
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VOL. LIII JANUARY, 1943. No. 1

THE CRICOPHARYNGEAL SPHINCTER— A ROENTGENOLOGIC STUDY.*

DR. FREDERIC E. TEMPLETON and DR. RICHARD A. KREDEL, Chicago.

The cricopharyngeus is a muscle which otolaryngologists regularly encounter during the passage of an esophagoscope. Exact conclusions concerning the appearance or action of this muscle cannot be drawn from endoscopic observations, however. Only the presenting surface and not the entire sphincter is seen. The presence of the esophagoscope is an abnormal situation which might give rise to abnormal appearances and actions.

Refinements in radiographic equipment and technique make it possible to study the physiology of the cricopharyngeus by a totally different method. These newer observations support the endoscopic opinions that the cricopharyngeus possesses the function of localized contraction and is in every sense a true sphincter.

The Roentgenologic Examination: The Roentgenologic examination is carried out by using a standard filming fluoroscope or "spot" machine, an instrument designed to record fluoroscopic images on films. It is possible, with this machine, to insert a film in front of the fluorescent screen and to change the milliamperage and kilovoltage from that used for fluoroscopy to that used for filming at any desired moment. Since this instrument is manufactured by all leading companies dealing in radiologic equipment and is found in many radiologic departments, no further description of the apparatus is necessary. The illustrations reproduced in this paper

*From the Division of Roentgenology, University of Chicago.

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are all made with such an instrument and represent selected stages of a fluoroscopic examination.

The patient, who needs no special preparation for the examination, is placed behind the fluoroscopic screen in one of the lateral positions. He is requested to hold about an ounce of a mixture made up of equal parts of barium and water in his mouth. The screen is centered over the pharynx, the chin elevated and the shoulders pulled backward and downward so as to clear the field. The patient is asked to swallow the entire bolus in one motion and not in a piecemeal fashion, for the bolus must be sufficiently large to distend the pharynx. This portion of the examination is termed the "active stage."

After the bolus passes through the pharynx, some of the barium mixture usually clings to the mucosa and outlines the collapsed lumen of the pharynx and esophagus. The patient is cautioned not to follow the first act with repeated acts of deglutition, for this eliminates the shadows of the collapsed lumen by forcing the remaining barium down the esophagus. This portion of the examination is called the "resting stage."

After the pharynx is observed in the resting stage the patient is asked to take a deep breath and close his mouth, pinch his nose and blow against the closed mouth and nose. The procedure is clarified by asking the patient to blow out his cheeks as if he were blowing into a horn, or to "blow your ears full of air." This is the Valsalva test performed against the closed mouth and nares instead of against the closed glottis, the conventional method of performing the exercise.

The patient is now turned into the anteroposterior position and the entire procedure repeated.

Films are exposed during the different stages of the examination. No difficulty is experienced in obtaining views of the resting stage and during the performance of the Valsalva test as the pharyngeal structures are quiescent.

Satisfactory views of the active stage cannot be obtained unless some method is used to synchronize the exposures with the act of deglutition. Laurell[1] reports a procedure for obtaining views of the pharynx distended with barium. He has the patient fill his mouth with barium and, with the

fluoroscope turned on, counts slowly and evenly. At the count of three the patient swallows. Laurell continues to count until the barium fills the pharynx. He notices the count at which this occurs — let us say the number is six. He repeats the procedure after bringing the film into a position for the exposure and with the fluoroscope turned off. On three the patient swallows and on six the exposure is made. This method depends upon close co-operation between the physician and the patient and often several trials are necessary before satisfactory views are obtained. Particular difficulty is experienced in patients who, because of dysphagia, cannot initiate an act of deglutition at will. The interval between the first attempt at deglutition and the passage of the bolus often varies considerably.

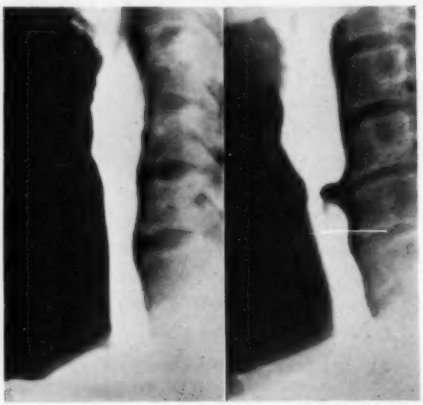
We have devised a second method which, after some practice, results in adequate views on almost every trial even though the patient experiences difficulty in swallowing. After the fluoroscopic observation, the patient is given about an ounce of barium mixture and asked to hold the material in his mouth. The fluoroscope is centered over the pharynx, turned off and a film brought into place. The examiner places the tip of his index finger on the tip of the patient's thyroid cartilage. The patient swallows. When the thyroid cartilage begins its upward movement, the finger is quickly withdrawn from the field and the exposure made.

The latter method is based on the observation that the bolus passes through the pharynx at the time the thyroid cartilage reaches its uppermost travel, during the act of deglutition. The interval between the origin and completion of the upward movement of the larynx is sufficiently long for the examiner to withdraw his finger from the field. As is also the case in Laurell's method, the success of the attempt is judged by observing the image as it flashes on the fluoroscopic screen during the exposing of the film.

The Normal Pharynx and Cricopharyngeal Region: Active Stage — In the active stage of deglutition the barium which distends the pharynx casts a silhouette of the lumen. In the lateral projection the posterior margin of the silhouette is wave-like and in some patients a pronounced indentation also occurs at about the level of the cricopharyngeus muscle. The anterior wall is irregular at the level of the laryngeal aper-

ture and sometimes at the level of the cricoid cartilage. Except for these variations, the anterior wall of the pharynx is straight and sharply defined (see Figs. 1 and 7).

In the anteroposterior projections the walls of the pharynx converge on the mouth of the esophagus. If the patient swallows a large bolus, the walls of the laryngeal pharynx distend and no structures are seen within the substance of the barium mixture. If, however, the patient takes a smaller swallow, the barium splits and passes down both pyriform sinuses,



A B

Fig. 1. The pharynx during the active stage of deglutition — lateral projection.

In both (A) and (B) the barium completely distends the pharynx. The irregular margin of the barium on the upper anterior walls is caused by the contracted laryngeal aperture. In (B) a sharp indentation along the middle of the posterior margin of the barium column probably represents the cricopharyngeus muscle. This indentation is frequently seen in patients who do not complain of dysphagia. The pulling-in of the anterior wall opposite the indentation is probably caused by the cricoid cartilage.

which are separated by a rounded, inverted-triangular, translucent shadow. This latter shadow represents the opposed walls of the central portion of the pharynx (see Fig. 2).

Resting Stage — After the larynx drops to its resting position, a thin coating of barium often clings to the mucosa and outlines the lumen of the pharynx and the upper esophagus. The valleculae and pyriform sinuses are clearly seen. The lateral margins of the pyriform sinuses are indented by the thyroid cartilages. This relationship is evident if these cartilages are calcified (see Fig. 3A). In the lateral projection the barium in the collapsed lumen of the lower laryngeal

pharynx continues on into the esophagus in a straight or slightly wavy line (see Fig. 4A).

Valsalva Test — Jönssen[2] in 1934 recorded the appearance of the pharynx during the Valsalva test performed as we described it earlier, using only air as a contrast medium. He devised this method of examination to show polypoid masses arising from the wall of the pharynx. The increased intrathoracic pressure set up by the Valsalva test is transmitted to the trachea and distends the pharynx. Intrapharyngeal masses stand out against the increased volume of air.

If the Valsalva test is performed after the passage of a bolus of barium mixture, the barium clinging to the walls of the pharyngeal and esophageal mucosa outlines those por-

A B C

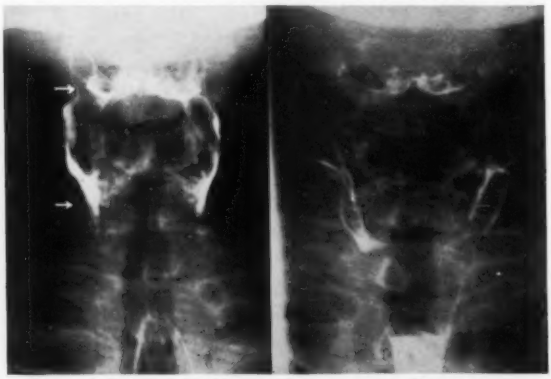
Fig. 2. The pharynx during the active stage of deglutition — anteroposterior projection.

In all three views a bolus of barium distends the pharynx and upper end of esophagus. The lumen at the lower end of the pharynx varies in width. In (B) the bulk of the bolus completely distends the walls so that none of the structures in the pharynx can be identified. In (A) and (C), particularly (C), the bolus is not large enough to completely distend the pharynx. Most of the barium passes down the pyriform sinuses. The central triangular shadow of radiolucency represents opposed portions of the pharyngeal walls.

tions of the lumen not distended with air. The effect that the Valsalva test has on the undistended portions of the lumen can be studied. In the frontal projection the pyriform sinuses balloon out but are held from overdistention by the thyroid cartilage. The pharyngeal walls above the level of the cartilages bulge laterally, forming two ear-like pouches, one on

either side of the pharynx (see Fig. 3B). In some patients enough barium remains in the pharynx and upper esophagus and outlines the mucosal folds. When this happens, the width of the lumen in the region of the cricopharyngeus can be measured. It varies from about 0.5 to 1 cm. in width and is much wider than that observed in the lateral projection (see Figs. 5A and 5C).

In the lateral projection the ear-like pouches extend forward over the thyroid cartilage. The anteroposterior diameter of the laryngeal pharynx is increased. At the level of the cricopharyngeal muscle, a heavy, soft tissue shadow, meas-



A B

Fig. 3. Pharynx during the resting stage and during the Valsalva test—anteroposterior projection.

(A) Resting stage. Small amounts of barium outline the lumen of the pharynx. The right vallecula is indicated by the upper arrow and the right pyriform sinus by the lower arrow. The calcified wings of the thyroid cartilage lateral to the pyriform sinuses can be seen.

(B) With the Valsalva test that portion of the pharynx above the level of the thyroid cartilage balloons much more than that portion contained within the wings of the thyroid cartilage.

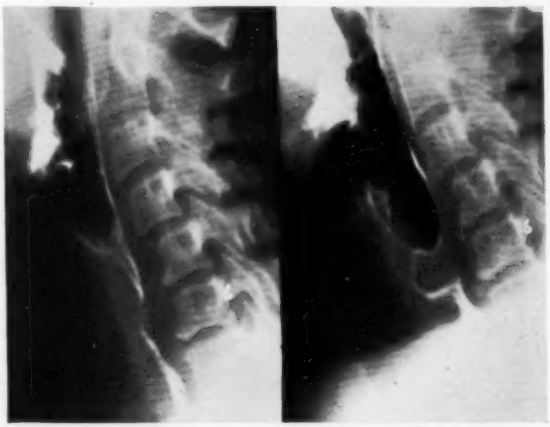
uring about 1 cm. in depth and 1 cm. in width, protrudes into the lumen (see Fig. 4B). The more vigorously the test is performed, the more prominent the shadow becomes. The lumen which curves about this shadow is narrow and appears to be pressed against the cricoid cartilage. The esophagus immediately below the mass distends slightly. The distention is particularly marked if large amounts of air are trapped in the esophagus.

This phenomenon is seen in all patients able to perform the Valsalva test properly. In no instance have we observed air

forced through the sphincter from the distended pharynx. When the patient ceases to perform the test the mass disappears, the lumen appearing as it did before the test was initiated.

Discussion: We believe that the structure sometimes seen along the posterior wall of the filled pharynx (see Fig. 1B) and which occurs during the performance of the Valsalva test (see Fig. 4B) is the cricopharyngeal muscle, for the following reasons:

1. The structure lies at the level of the cricopharyngeal muscle and pulls in the walls of the pharyngoesophageal junc-



A B

Fig. 4. Pharynx during the resting stage and during the Valsalva test — lateral projection.

(A) Resting stage. The upper portion of the pharynx is distended with air but the lower portion is contracted. Note the slight anterior curve of the upper esophageal lumen opposite the sixth cervical vertebra.

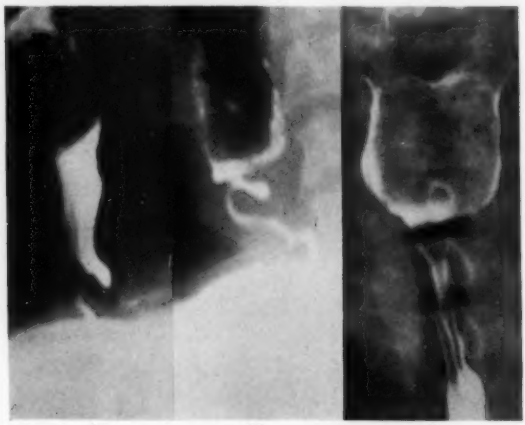
(B) During the Valsalva test. The collapsed inferior portion of the pharynx distends and the anterior curvature of the upper esophageal lumen becomes more pronounced. It is believed that the increased curving of the collapsed lumen below the pharynx is caused by increased contraction of the cricopharyngeus muscle. This phenomenon is a regular occurrence in all patients able to perform the Valsalva test.

tion in a manner one would expect when the cricopharyngeal muscle contracts. From the anatomical make-up, the contracted cricopharyngeal muscle should pull the lumen forward against the cricoid cartilage. The thin lumen, seen in the lateral, and the broad lumen, seen in the frontal projection, substantiate this assumption.

2. The muscle bundle has a distinct relationship to the

posterior pharyngeal diverticulum. In three cases of small, asymptomatic, pharyngeal diverticula and in three cases of large pharyngeal diverticula the mouths of the diverticula lie in the center of the muscle bundles (see Fig. 5). The dissections of Killian[3] indicate that the posterior pharyngeal diverticula protrude between the oblique and lateral fibres of the cricopharyngeal muscle. It is suggested that the part of the bundle above the mouth of the diverticulum represents the oblique and the part below the circular fibres.

3. The curved appearance that the lumen immediately below the pharynx assumes during the performance of the



A B C

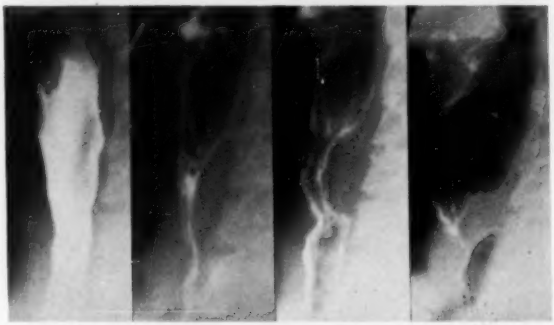
Fig. 5. Small posterior pharyngeal diverticulum.
(A) Resting stage — lateral projection.
(B) During the performance of Valsalva test — lateral projection.
(C) Resting stage — anteroposterior projection.
In (B) a small diverticulum protrudes backward and lies within the mass of tissue which forces the lumen below the pharynx anteriorly. In (C) the width of the lumen immediately below the pharynx is wider than that observed immediately below the diverticulum shown in the lateral projection (A).

Valsalva test cannot be attributed to simple kinking caused by the increased pressure forcing the pharynx downward. When the hyoid bone is related to the spine, no significant change in its position occurs during the performance of the Valsalva test. Furthermore, air is not forced from the pharynx into the esophagus. It does not seem likely that sim-

ple kinking of the lumen could resist the pressure set up by the Valsalva test.*

Additional evidence that kinking does not occur is offered in a case observed after laryngectomy. The muscle bundle was demonstrated with the patient performing the Valsalva test against the closed opening of the tracheotomy tube. In this latter instance it was not possible for kinking to occur, since air pressures in the trachea could not be transmitted to the pharynx (see Fig. 6).

These observations support Negus'[4] contention that an important function of the sphincter is to prevent air from



A B C D

Fig. 6. Pharynx after laryngectomy.
(A) Active stage.
(B) Resting stage.
(C) During esophageal speech.
(D) During performance of Valsalva test. The Valsalva test was performed with the patient attempting to exhale after covering his tracheotomy opening with his finger. During phonation a broad mass behind the lower portion of the junction of the pharynx and esophagus is seen. It is believed that this represents the organ which vibrates when the patient produces his tone for esophageal speech. During the Valsalva test the mass becomes more prominent and narrows the lumen between the pharynx above and esophagus below. It is believed that the mass represents the contracted cricopharyngeus muscle.

entering the esophagus during a respiratory cycle where it would serve no useful purpose and detract much from the tidal air volume.

The nervous control of the cricopharyngeus is not thor-

*During the Valsalva test, performed against a closed mouth and nose, the intrathoracic pressure is about equal to the intrapharyngeal pressure. If fluid or air become trapped in a relaxed esophagus, the increased pressure forces the contents against the cricopharyngeal sphincter (see Fig. 6D). It can, therefore, be argued that a flow of air from the pharynx into the esophagus is not possible even in the absence of a sphincter since pressures in each organ are approximately equal. This argument does not explain why the lumen through the cricopharyngeal region fails to distend when these pressures are exerted (see Fig. 4B). Obliteration of the lumen by a muscle in a state of contraction sufficient to resist the pressures is a much more logical explanation.

oughly understood. Although its action blends with that of the adjacent musculature during an act of deglutition, the independent action it displays, particularly during the Valsalva test, subjects it to special study. The force which propels a bolus thru the laryngeal pharynx is produced by a peristaltic wave beginning with a diffuse contraction of the oropharynx and progressing in an unbroken manner through the laryngeal pharynx, past the cricopharyngeal muscle and down the esophagus. This wave is preceded by a wave of inhibition which allows the lumen ahead of the wave of con-

A B C

Fig. 7. Serial views showing pharyngeal peristalsis during the active stage of deglutition.

(A) Early in the act. The pharynx and upper end of esophagus are completely distended. The wavy appearance of the posterior wall is caused by pressure from the intervertebral discs.

(B) Peristaltic wave which began high in the pharynx has narrowed the extreme upper portion of the pharynx.

(C) The peristaltic wave has progressed so that now the entire pharynx is in the state of contraction.

traction to be distended by the bolus. Physiologists term this wave "primary peristalsis" and are agreed that the harmonious sequence of contractions involved in the act of deglutition depends upon an extrinsic nervous mechanism and not on a simple conduction of the wave from one portion of the wall to the next. Since the inhibitory wave extends far ahead of the contraction wave, fluids taken when the patient stands drop quickly through the pharynx and upper portion of the esophagus. The propelling force is gravity. When the force of gravity is neutralized by carrying out the examination

with the patient in a horizontal position or by using thick mixtures, the peristaltic wave is easily demonstrated at Roentgen examination (see Fig. 7).

We agree with Negus,[4] Pancoast, Pendergrass and Schaeffer[5] that there is no need to postulate an active expansion of the lumen creating a negative pressure which sucks the bolus along as advocated by Barclay.[6] We have not observed this and fail to see how it can occur. There is nothing in the attachment of the musculature which would give rise to active expansion of the lumen with muscular contraction. Distention of the lumen can only occur with muscular relaxation and positive pressure in the lumen.

If the passage of a bolus during the act of deglutition depends solely on a harmonious sequence of contraction waves preceded by inhibitory waves, we may postulate that the mechanism suggested by Thomas[7] for the pyloric sphincter plays a rôle in the opening of the cricopharyngeal sphincter. Thomas contends that gastric evacuation occurs whenever the intragastric pressure near the pylorus exceeds the resistance of the sphincter.

The cricopharyngeal sphincter is normally closed. The term "closed" does not necessarily connote active muscular contraction. The lumen of a flaccid tube remains obliterated if the lumen is not occupied by some substance. It is supposed, however, that the cricopharyngeal sphincter is held closed against changes in pressure occurring in the pharynx during respiration, coughing, blowing, etc., by the normal tone existent in striated muscle, probably augmented by additional contraction stimulated by certain nervous reflexes. The action of the cricopharyngeus during the performance of the Valsalva test suggests this.

During normal deglutition the inhibitory wave is strong enough to neutralize any impulses causing muscular contraction. This decreases the resistance which the muscle offers to the passage of a bolus. In normal individuals the force exerted on the bolus by the peristaltic wave is great enough to overcome any resistance the sphincter offers. As a consequence, passive opening of the sphincter occurs. It appears that the opening of the cricopharyngeus may be only an expression of high intrapharyngeal (or esophageal in the case of belching) pressure and that a closed pharyngeus is only an expression of low pharyngeal or esophageal pressure.

If the passage of a bolus depends on the force set up by a peristaltic wave, any marked weakness in the pharyngeal musculature will fail to force a bolus through a normally closed sphincter. We are not, from Roentgenologic observations, justified in using the term "cricopharyngeal spasm" since it is not possible to determine the amount of pressure set up in a pharynx in a patient whose cricopharyngeal muscle fails to open adequately. This phenomenon is regularly observed in bulbar palsies and in myasthenia gravis.

Whether abnormal contraction of the cricopharyngeus sufficient to resist the powerful pharyngeal contractions of a normal pharynx exists is not known. Perhaps this condition occasionally gives rise to the acute pain sometimes experienced when one attempts to swallow an unusually large amount of fluid at once. It is advocated as a cause of globus hystericus by Antoni[8] but we have examined a number of these patients and in none have we observed abnormal action of the pharyngeal muscle and sphincter even though some of the patients complained of symptoms at the time of the examination.

CONCLUSIONS.

The cricopharyngeus muscle is a true sphincter which can be easily demonstrated at Roentgenologic examination.

When the Valsalva test is performed, the contraction of the cricopharyngeus is intensified.

BIBLIOGRAPHY.

1. Laurell, H.: Zur Frage der Entstehung kardianaher Magendivertikel und Zenkerscher Oesophagusdivertikel. Zugleich ein Beitrag zum Schluckmechanismus. *Acta Radiol.*, 12:455-478, 1931.

2. Jönsson, G.: Röntgenological Findings in Malignant Tumors of the Nasopharynx. *Acta Radiol.*, 15:1-7, 1934.

3. Killian, G.: Quoted by Barclay, A. E., in "The Digestive Tract." Cambridge Univ. Press, 1933.

4. Negus, V. E.: Affections of the Cricopharyngeal Fold. The Laryngoscope, 48:847-858, Dec., 1938.

5. Pancoast, H. K.; Pendergrass, E. P., and Schaeffer, J. P.: The Head and Neck in Roentgen Diagnosis. Charles C. Thomas, Springfield, Ill., 1940.

6. Barclay, A. E.: Normal Mechanism of Swallowing. *Brit. Jour. Radiol.*, 3:534-546, 1930.

7. Thomas, J. E.: The Mechanism of Gastric Evacuation. *Jour. A. M. A.*, 97:1663-1668, 1938.

8. Antoni, N. R. E.: Paralysis of the Pharynx. *Acta Med. Scandinav.*, 59:745-776, 1923.

COMPOSITE POSTOPERATIVE THERAPY FOR THE LARYNGECTOMIZED.*

DR. JAMES SONNETT GREENE, New York.

As you know, the first successful laryngectomy was performed by Billroth[1] in 1873. Since that time improvements in technique have greatly diminished operative mortality and postoperative complications.

Fortunately, concomitant with these improvements in surgical procedure, methods of re-education have been developed to restore some measure of voice and speech to the patient who has lost his larynx. These re-educational methods involve either *1.* the use of other anatomical structures to produce voice, or *2.* the use of the mechanical larynx.

Recent experiments in the physics of voice production[2] have shown that in vocalization the air stream rising from the lungs is broken up, as it passes through the larynx, into a train of "puffs" or pulses by the vibrating vocal cords.

These interrupted puffs function as sound initiators. That is, when they encounter the relatively static supraglottal air they create a train of sound waves which, passing through the speech molds, are transformed into audible speech; therefore, to produce voice-using anatomical structures other than the larynx, two things are required: a column of air and some closely approximated tissues which can be set in vibration.

The column of air may arise from various sources and may be: *1.* buccal, *2.* pharyngeal, *3.* esophageal, or *4.* gastric.[3] Best results are obtained, however, by using the esophagus, into which the air is swallowed or aspirated, held for a moment, then slowly expelled past the closely approximated vibrating tissues, which can be termed the pseudoglottis.

The pseudoglottis may be formed by a variety of tissues: the tongue against the posterior pharyngeal wall, the pillars

*Read at the meeting of the New York Academy of Medicine, Section on Otolaryngology, May 21, 1941.

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of the fauces, adventitious folds of the hypopharynx, the anterior wall, the collapse of one esophageal wall against the other to produce a pseudosyrinx, a cicatricial band against normal tissue, a pair of cicatricial bands, a laryngeal tonsil,[4] etc.

In developing a substitute voice, the patient must learn to co-ordinate the processes of respiration, aerophagia and phonation. This, of course, is a difficult procedure. The patient's first tendency is to swallow large quantities of air, and the subsequent eructation causes his speech to have a marked belch-like quality; however, he later learns to aspirate the air unconsciously, by an involuntary contraction of the sternohyoid, thyrohyoid, genohyoid, mylohyoid and anterior portion of the digastric muscles.[5] As he learns to do this, the flow of words gradually becomes smoother.

Of course, there are a number of patients in whom it is impossible, for one reason or another, to develop a substitute voice. In these cases the mechanical larynx is indicated.

The first such mechanical larynx was devised, apparently, by Czermak in 1859 for a patient with a complete chronic laryngeal stenosis, and since then these instruments have been modified and improved by a number of investigators, including Czerny, Eiselberg, von Bruns, Gottstein, Sorenson, Onodi, Hinajav, MacKenty, Fletcher and Wegel.[6]

Their instruments were all constructed on the same basic principle: a tube containing a reed was connected to the laryngectomized patient's tracheal fistula and led to the mouth. The current of air expired from the tracheal fistula set the reed in vibration, thereby producing sound which was articulated by the speech molds and converted into speech.

Variations in these instruments included production of the air current by some form of bellows or pump activated by the hand, arm or thoracic wall; however, all such mechanical larynges have certain obvious disadvantages: They are conspicuous; the voice is unnatural and has a "quack-like" quality; and salivation is excessive. Also, some authorities feel that the continued irritation of the throat-piece in the tracheotomic fistula may give rise to new cancerous growths.[7]

In addition, there is a definite hygienic factor involved.

The patient usually carries the artificial larynx in his pocket or, if a woman, in her handbag. Since the device is continually being used and returned to the pocket or bag, there is no way to insure the cleanliness of the rubber tube. In this connection it must be remembered that the patient cannot cough out any unnatural matter that may be introduced into his mouth by the tube. But perhaps more serious is the danger of infection at the tracheal opening. Here there is a foreign body, a cannula, which might serve as a fertile medium for foci of infection.

There has been, therefore, a definite need for a simple device which would not only do away with the more obvious disadvantages of the old-type mechanical larynx, but which would also obviate any possibility of infection. The ideal instrument would be one that could be used externally.

Such a device has recently been developed by Gilbert Wright for the National Hospital for Speech Disorders. We are about to furnish patients, who cannot master esophageal speech, with this new type of mechanical larynx — the Sonovox.

This instrument consists of a small capsule containing two battery-connected coils, over which a diaphragm is placed. When the capsule is held over the glottal region, electrical contact is established and the diaphragm vibrates. The vibration is communicated, through the tissues of the neck, to the static air in the hypopharynx, setting the air in vibration and initiating sound waves which are then transformed into speech via the speech molds.

The instrument is completely external, is inconspicuous and precludes all possibility of infection or tracheotomic irritation.

Here is a patient who has developed an esophageal voice, another patient who uses the old-type mechanical device, and a patient who uses the Sonovox. I want to emphasize the fact that postoperative treatment of the laryngectomized patient should not be limited solely to re-education.

Such a patient has usually led a full, active life until the age of 40, 50 or 60 years, when he suddenly finds himself cut

off from employment and normal social intercourse because of the removal of his larynx and the consequent loss of voice. The psychic trauma induced by this sudden change in economic and social status is often as serious as the physical trauma itself; therefore, postoperative therapy for these patients should embody psychological as well as re-educational measures, and its aim should be not only the restoration of the patient's voice to as nearly normal as possible, but also the readjustment of his mental attitude to his changed status.

In this connection, group therapy is invaluable. It brings the new patient in contact with advanced patients who serve as examples and sources of encouragement. Thus, the patient realizes that his position, while unfortunate, is not unique, and he is able to practice developing his new voice in a sympathetic milieu — a milieu in which everyone is in a similar situation.

REFERENCES.

1. Morrison, W. Wallace: The Production of Voice and Speech Following Total Laryngectomy. *Arch. Otolaryngol.*, 14:4, Oct., 1931.

2. Carhart, T. Raymond: Air-Flow Through the Larynx. *Quar. Jour. Speech*, Dec., 1940.

3. Jackson, C. J., and Jackson, C. L.: The Larynx and Its Diseases. 1937.

4. *Ibid.*

5. *Ibid.*

6. Morrison, W. Wallace: Opus Cit.

7. *Ibid.*

8. Schall, Leroy A.: The Psychology of Laryngectomized Patients. *Arch. Otolaryngol.*, 28:4, Oct., 1938.

A CASE OF UNILATERAL OTOSCLEROSIS WITH A MICROSCOPIC DESCRIPTION OF THE FOCUS.*†

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In spite of extensive work on otosclerosis, including many microscopic studies, the etiology of this disease is not clear and the details of the pathology which is involved has remained controversial. Because of this, each single case which is reported contributes some details to the explanation of the condition.

In this paper the writer intends to discuss a case of otosclerosis which he detected in a patient whose temporal bones were studied microscopically for other reasons.

Short History of the Case: The male patient, 52 years of age, died of an acute meningitis after the onset of an acute otitis media of the right ear. According to the reports of relatives and friends, among them two physicians, he had never experienced previous ear troubles and his hearing had been essentially normal. Hearing was apparently normal in the patient's left ear during the course of the infection, which was localized in the right ear. Because of his general condition, the writer refrained from making a hearing test of the left ear.

At the autopsy both temporal bones were prepared for microscopical investigation in the following manner: fixation in 10 per cent formaldehyde; decalcification in 5 per cent nitric acid; dehydration and hardening in 80 per cent absolute alcohol; embedding in 2-15 per cent celloidin; sections at 20 micra; each tenth section of the petrous apex, each third section of the inner ear and each fifth section of the middle ear were stained in hematoxylin-eosin.

A description of the microscopical findings in both temporal bones was given in an earlier paper by the writer, except for the otosclerotic focus in the left temporal bone, the presence of which was mentioned briefly.[1] In the present

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paper interest is centered on the left temporal bone where an otosclerotic focus was found.

Extension of the Focus: The posterior end lies between the facial canal and the ampullae of the anterior and horizontal canals (see Fig. 1). A few sections forward the focus expands, gradually encompassing the space between the maculae and the facial canal (see Fig. 2). It approaches the upper anterior wall of the oval window lying between the macula cribrosa utriculi and facial canal, with its margins immediately at the endosteum of the former structure and at the periosteum of the latter structure (see Fig. 3).

The pathological bone surrounds the anterior half of the fenestra and approaches the junction of the vestibulum with the scala vestibuli where it borders the upper two-thirds of this latter structure. Further anteriorly, the focus is bordered medially by the macula cribrosa utriculi, vestibulum and upper part of the scala vestibuli of the basal turn; laterally and above by the canal of the musculus tensor tympani and of the facial nerve. Still further anteriorly, the focus lies between the facial canal, the musculus tensor tympani and the cochlea, enveloping the lateral side of the middle and apical turn. Finally, the focus ends as a wedge-shaped protrusion between the middle and the apical turn on the lateral side of those structures (see Fig. 10).

The estimated dimensions of the focus are anterior-posterior, 2.6 mm.; lateral-medial, 2.5 mm.; and in the vertical direction, 4.5 mm.

The Nature of the Focus: The focus shows three distinct types of tissue: *a.* a soft form in which osteoplastic bone marrow predominates; *b.* a sclerotic form with compact irregular bone; *c.* a highly vascularized form with many blood vessels and blood spaces. These three forms are not sharply delineated.[2]

In its anterior portion, where the focus envelopes the cochlea, osteoplastic bone marrow forms the dominant picture (see Fig. 10). While further posteriorly, close to the canal of the musculus tensor tympani, many large blood vessels are present. More medialward the focus changes into the soft type (see Fig. 8), and most medialward into the compact

form. In the region posterior to the fissula ante fenestram the focus is compact, with large engorged blood vessels (see Figs. 3 and 4). Laterally, the focus passes through a stage of compact bone (see Figs. 5 and 6) and ends as soft fibrous

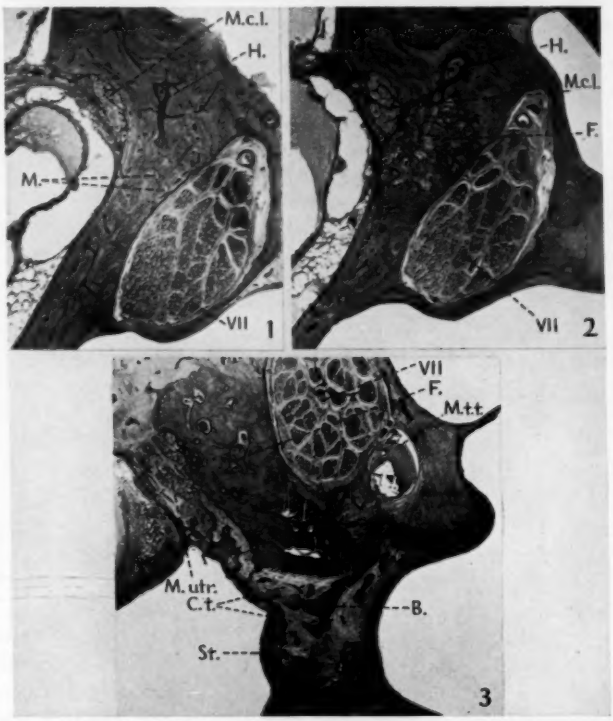


Fig. 1. (H) Haversian system. (M) Newly formed marrow spaces along an Haversian canal. (VII) Nervus facialis. (M.c.l.) Macula cribrosa ampullae lateralis.

Fig. 2. (H) Haversian canal. (F) Focus of soft type. (VII) Nervus facialis. (M.c.l.) Macula cribrosa ampullae lateralis.

Fig. 3. (F) Focus of compact type. (St) Deformed footplate of the stapes. (B) Bone trabeculae in the upper anterior margin of the oval window. (Ct) Connective tissue. (VII) Nervus facialis. (M.utr.) Macula cribrosa utriculi.

material between the horizontal facial canal and the macula (see Fig. 2).

Of special interest is the region of the fissula ante fenestram. It is formed of dense connective tissue with many

irregular bony trabeculae. Cartilage cells are not present (see Figs. 6 and 7). The whole anterior third of the niche of the oval window is occupied by this type of connective tissue which is continuous with the anterior part of the annular ligament and with the connective tissue anterior to the oval window (see Fig. 4).

In general, one might say that at its posterior and anterior ends the focus has the softer form of bone with many marrow spaces, while in the center compact bone prevails. In the region of the fissula ante fenestram and in the anterior third of the oval window the focus presents a remarkable accumulation of dense connective tissue with many bone trabeculae. One has the impression that the center represents the older bone, apparently the origin of growth, with the deposition of younger bone expanding anteriorly and posteriorly.

Histology of the Focus: In the most posterior part, between the facial canal and the ampulla lateralis there is a cross-section of an Haversian canal from which branches spread in various directions; one of these branches runs downward parallel to the capsule giving off branches laterally and medially (see Fig. 1). The main canal shows in its center a blood vessel imbedded in loose connective tissue with spindle-shaped cells. The whole structure is wrapped in a dark blue-stained wall which contains very dark lines. On the outside of this canal, layers of lamellar bone are present. The darker stained areas in the walls of the canal seem to be an accumulation of calcareous material which is densest in the black lines. Schaffer[3] believes that the walls of these blood vessels consist of a very resistant precollagenous substance, the intensive staining reaction being possibly due to calcification. Most of these branches, like most, if not all, of the smaller Haversian canals, are filled with calcareous material in which one can distinguish, with high magnification, a few fibres of connective tissue, but no blood vessels. The big descending branch is interrupted by newly formed darker stained irregular bone which seem to follow the course of the vessel. The black lines disappear as if dissolved, the calcareous material so released presumably is available for the formation of new bone.

At the dorsal point in this region, where the focus either originated from the vessel or enters it, there is a small globe-

shaped, deeper pink-stained area which consists of irregular lamellae in an homogeneous mass and of lighter spaces containing nucleated cells. These seem to be osteoblasts which

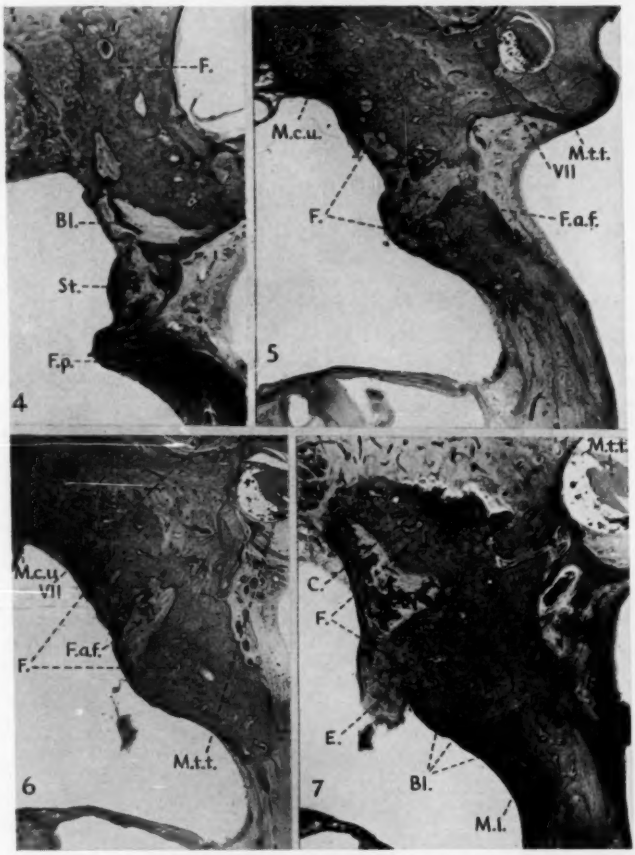
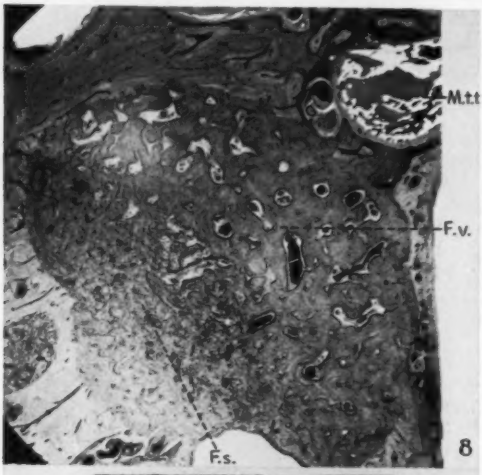


Fig. 4. (F) Focus. (St.) Stapes. (Bl) Blood vessel. (F.P.) Focus in promontorium. (VII) Facial canal (nerve torn out in sectioning).

Fig. 5. (F) Otosclerotic focus. (F.a.f.) Region of fissula ante fenestram. (VII) Nervus facialis. (M.c.u.) Macula cribrosa utriculi (F.a.f.) Connective tissue and newly formed bone in the region of the fissula ante fenestram.

Fig. 6. (F) Otosclerotic focus. (F.a.f.) Region of fissula ante fenestram. (M.c.u.) Macula cribrosa utriculi. (VII) Facial nerve. (Mtt) Musculus tensor tympani. (V) Vestibulum.

Fig. 7. (F) Focus. (E) Exostosis. (Bl) Engorged blood vessels. (C) Cleft between focus and normal bone (artificial). (Ml) Highly vascularized mucous layer. (Mtt) Musculus tensor tympani.



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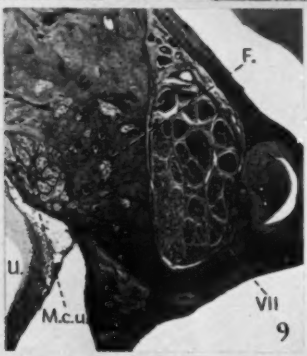

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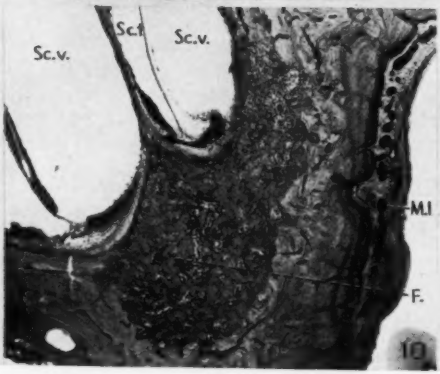

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change into osteocytes. Thus is formed a structure which Manasse has called "sieve-like bone" (see Fig. 1). Actually, the canal seems to send out marrow spaces in all directions along which the formation of new bone takes place. Downward the focus continues as a narrow strip and becomes continuous with two marrow spaces which are interconnected and finally ends in the canal as a rounded tip. About 60 micra forward, many marrow spaces, separated from each other by "sieve-like bone" trabeculae, are present. Some giant cells, osteoblasts, fibroblasts, etc., are to be seen in these marrow spaces, the osteoblasts being arranged in rows or scattered at random. Some of these marrow spaces occupy the areas where Haversian canals had been present and give rise to branches which are interconnected (see Fig. 2).

A connection between the marrow spaces and the endosteal layer of the facial canal was demonstrated. Fairly large canals perforate the bony wall, carrying connective tissue, spindle-shaped cells and a blood vessel in the center. Immediately after entering the bone they spread in all directions, forming larger marrow spaces which contain numerous osteoblasts arranged in rows (see Fig. 2).

About 200 micra anterior, the focus in its upper part shows many marrow spaces separated from each other by wavy trabeculae running in all directions. In the lower part there are no marrow spaces and the bone has a more compact structure (see Fig. 8). Where the focus meets with an obliterated Haversian canal it forms a round exostosis and follows the course of the canal, dissolving its blue-stained walls (see Figs. 7 and 8).

The posterior half of the footplate is rich in cartilage, which is completely absent in the anterior half. In the upper frame of the oval window is a layer of dense connective tissue with a big blood vessel. This island of connective tissue is imbedded in the newly formed bone (see Fig. 4). In the upper part marrow spaces prevail. More anteriorly, the upper

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Fig. 8. (Fs) Soft type of the focus. (Fv) Vascularized type of the focus. (Mtt) Musculus tensor tympani.

Fig. 9. (F) Focus of soft type with some narrow spaces. (Ct.) Connective tissue in upper anterior margin of oval window. (M.cu.) Macula cribrosa utriculi. (VII) Nervus facialis. (U) Utriculus.

Fig. 10. (F) Soft type of the focus. (Scv) Scala vestibuli. (Sct) Scala tympani. (ML) Highly vascularized mucous layer.

part of the footplate contains some marrow spaces and is thickened, with an exostosis lateralward. The anterior crus likewise is thickened and consists of irregular bone.[4]

In the marrow spaces of the upper part of the focus some giant cells are present. A large canal, containing a blood vessel and loose connective tissue with many cells, passes from the endosteum of the canal of the musculus tensor tympani into the focus and forms a few marrow spaces (see Fig. 7).

More anteriorly the footplate is markedly thickened, convex towards the vestibulum and consists of irregular wavy lamellae. The edges are serrated and the stapes is imbedded in connective tissue. Several large blood vessels and canals which are connected with the facial canal and with the mucous layer of the oval niche are present in this area (see Fig. 4).

Above the anterior part of the stapes the connective tissue is less dense and contains large engorged blood vessels. Some trabeculae of "web-like" bone are to be seen in the tissues (see Fig. 4). In the vestibular lining of the footplate a small engorged blood vessel is present.

The pathologic bone traverses the niche of the oval window in front and is continuous with similar bone below in the promontorium. The upper third of the promontorium is formed by compact irregular bone with many engorged blood vessels which anastomose with blood vessels in the mucous layer on the lateral side (see Fig. 4). The mucous layer covering the region of the focus is very much thickened and is highly vascularized. A large exostosis projects into the vestibulum at its junction with the scala vestibuli (see Fig. 7).

The bony trabeculae show a remarkable contrast with the bone in other parts of the focus. They are apparently developed directly from connective tissue while the latter is formed to a considerable extent from cartilage rests and by a remodeling of old bone.

More anteriorly, the focus contains in its lower part, that is, at the promontorium, more marrow spaces and follows the course of an Haversian canal parallel to the vestibular wall. In its upper part the focus is also in connection with an Haversian system (see Fig. 6).

Lange[3] observed that the otosclerotic focus seems to lie in a space in the labyrinthine capsule. In some of the microscopic sections one observes a cleft between the focus and the normal bone which follows exactly the contour of the focus. Even if such a cleft is considered as an artefact, it demonstrates the sharp demarcation of the focus (see Fig. 7).

Interglobular spaces are absent in the focus; they are transformed and utilized in the formation of new tissue. At some points dark-stained cement lines separate the focus from the adjacent normal bone.

Discussion: In this case we are dealing with a unilateral otosclerosis. This is rather exceptional since the disease generally is bilateral and usually symmetrical. It is not unusual to find a marked difference in the acuity of hearing in the two ears. In most cases one ear becomes involved first and shows a higher degree of deafness than the other. Furthermore, the writer, like many other authors, has observed cases with otosclerosis on one side while the other side retains normal hearing. The observance of this fact should not be regarded as evidence that this ear does not have an otosclerotic focus; it indicates only that this focus, if present, is either very small or is located in such a place as not to interfere with hearing. In our case, otosclerosis on the left side did not give subjective symptoms, but it is not certain that the patient would not have revealed objective symptoms which would have been apparent upon suitable test. It would be very surprising that an otosclerotic focus of such an extent as this would not have had at least a lengthened bone conduction and lateralization on the diseased side. The absence of subjective noises is also surprising; but we know that this symptom, if not very marked, is often neglected by the patient when he becomes accustomed to such noises. The same holds true for the hearing as long as it is only slightly impaired. In all likelihood such has been true in this case.

The focus is composed of a tissue which shows three different characteristic forms; a compact part composed principally of dense bone; a soft part containing many marrow spaces of different sizes; and a vascularized portion. These three forms are not sharply delimited and represent obviously different stages in the course of growth corresponding to

their age. The soft and vascularized portions represent successive phases in the development of the lesion, while the compact part is the oldest and presents the final condition of the otosclerotic focus.

The center of the focus, which in this particular case corresponds approximately to the region immediately posterior to the fissula ante fenestram, is composed chiefly of compact new bone representing the oldest part of the focus and therefore its point of origin; while the parts on its periphery are of the soft and vascularized form representing younger stages. The fissula itself is composed chiefly of dense connective tissue without cartilage rests.

The focus follows the course of blood vessels around which marrow spaces develop. Numerous cells in these marrow spaces change into osteoblasts and form new bone, while the cartilage islands and the old bone are destroyed.

The dark blue walls of those blood vessels, which in the course of normal development become narrowed, are dissolved and their contents of calcareous material may be utilized in the formation of bone.

Summary: The histological description of a case of unilateral otosclerosis is presented. The cause for the new growth seems to be related to the abnormal presence of blood vessels in this region. This new growth appears to pass through a phase of relative inactivity and then to accelerate gradually by a physiological or pathological stimulus involving the general circulatory system.

The case is further evidence for the occurrence of unilateral otosclerosis. It demonstrates, also, that such unilateral disease may exist unnoticed by the patient, but it does not exclude the possibility that objective symptoms of the disease might have been detected upon suitable test.

BIBLIOGRAPHY.

1. BENESI, O.: Zur Pathogenese d. Otitischen Fruehmeningitis. *Monatsschr. f. Ohrenheilk.*, 1935.

2. MAYER, O.: Untersuchungen über die Otosclerose. *Wien. u. Leipzig*, 1917.

3. SCHAFFER, J.: Lehrbuch d. Histologic u. Histogenese. Leipzig: Engelmann, 1922.

4. COVELL, W. P.: The Ossicles in Otosclerosis. *Acta oto-laryngol.*, Vol. 28.

5. LANGE, W.: Handbuch d. speciellen pathol. Anatomie u. Histologie. *Heinke u. Lubarsch.*, Vol. 12, 1926.

HEADACHE AND FACIAL NEURALGIA.— CLASSIFICATION AND DIFFERENTIAL DIAGNOSIS.*

DR. T. ROY GITTINS, Sioux City, Iowa.

The complaint of pain somewhere in the head or face is probably the most common symptom encountered in the everyday office practice of ophthalmology and otolaryngology and is quite commonly encountered in the practice of any physician. Symptoms associated with headache or face pain are so many and varied that the patients may, and commonly do, appear in the offices of the general physician, general surgeon or internist, or seek the advice of members of any of our special groups. Migraine and other types of vasomotor headache, with or without associated symptoms, influenced and propagated as they are by hereditary tendencies, have resisted valiantly any and all attempts at limitation, regimentation or control. For 50 or more years, responsibility for treatment has been shifted at intervals to new specialties, not because the members of these specialties were peculiarly equipped to handle the problems of diagnosis and treatment but rather because their enthusiasm over their new specialty gave them much confidence, and members of the older specialties much relief. Despite the fact that we have lived during the age of specialization, I hazard the thought that most of our patients come to us because we are physicians, regardless of our specialties. They naturally believe that our horizons are not limited by any artificial divisions of their anatomy or of our medical thinking.

Migraine, as the most constant in behavior and well known of the various types of periodic headache, is just as much a general disease complex as is asthma, rheumatism or gout. The symptomatology and clinical course of this very common condition has been described in literature for hundreds of years without any real changes, except perhaps that the clearest descriptions were written centuries ago. The best and most complete description of the symptomatology of this syndrome that I have in my files appeared in the literature in

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1797. A physician, from London, gave a paper on this subject before a group of physicians in New York. After giving a very complete description of the symptoms and stressing the lack of physical signs, he spent considerable time lauding his favorite treatment, which was the administration of from a gill to a pint of hard cider each morning before breakfast. Such treatment, if advocated in a paper at the present time, would naturally provoke amusement, but certainly it was much more agreeable to the patients than some of the ill-advised nasal, sinus and abdominal operations to which similar patients have been subjected during modern times.

The first specialty to assume some real responsibility for the treatment of migraine or other forms of vasomotor headache was that of ophthalmology, and the members of this group have struggled valiantly through the years to force this condition to respond to treatment with refractive lenses, prisms, muscle exercises, etc.; but the specialty that really walked in where more seasoned practitioners feared to tread was rhinology. Some 25 or 30 years ago it was rather suddenly and dramatically decided in many scattered centers that nasal obstructions, sinus infections and ganglion involvements were the root of all evil, especially if that evil was characterized by pain any place in the face or head, regardless of associated cortical symptoms, other general symptoms or a history of hereditary tendencies. I think we all admit that nasal and sinus surgery has been in disrepute for many years with general physicians and the general public. We must admit, too, that much of this disrepute came as a natural result of ill-advised nasal and sinus surgery; ill-advised because good scientific methods of handling acquired difficulties were sacrificed in a vain attempt to alter hereditary tendencies. Nasal and sinus surgical procedures have advanced rapidly to equal status with those of other specialties, when dealing with real pathology. Differential diagnosis has lagged farther behind the development of surgical technique and instrumentation in the special field of rhinology than in any other branch of medicine. Although at this time I am concerned mainly with vasomotor headache and neuralgia, I am not unmindful of the close family ties between these conditions and allergic and other forms of vasomotor rhinitis and sinusitis.

Periodic headache or facial neuralgia, especially if the symptoms are repeated over fairly long periods, with intervals of good health, with or without associated symptoms such as temporary blurred vision, vertigo, paresthesias, aphasia, nausea and vomiting, which appear in the ordinary ambulatory office patient, have usually a vasomotor basis of hereditary origin. By vasomotor basis I mean transient upsets in physiology, local tissue or fluid reactions in the brain, meninges, ganglions and nerve sheaths, with no real pathology which can be proven by physical examination, Roentgen or laboratory studies, operation or postmortem findings. I use this term "vasomotor" as an adjective when referring to certain periodic headaches, neuralgias and associated symptoms, for lack of a better descriptive term. Other adjectives, such as migrainous, allergic, endocrine, functional, myalgic, etc., refer to indefinite subdivisions of the main group; but as yet to my knowledge there is no appropriate name for this main group. I ask again for someone to suggest an appropriate name and thereby clear up considerable confusion. Such patients as are now being considered usually have periods of good health between their attacks and have ample time and opportunity to consult members of any or all branches of the medical profession, as well as members of the various cults. These patients are really not sick except during the attacks but there is stubborn resistance to any and all efforts directed toward prevention of recurrence of similar attacks, regardless of the type of treatment or operation. Such behavior in patients suffering from vasomotor headache or facial neuralgia, whether these conditions are classed as migrainous, allergic, endocrine or functional, has produced a real diagnostic problem throughout the years: the problem of treatment diagnosis versus differential diagnosis.

By *treatment diagnosis* I mean fitting the symptoms into the treatment possibilities of a particular specialty. For instance, a patient with unilateral frontal headache may be fitted with corrective lenses or prisms by the ophthalmologist, when the history clearly indicates that transient blurring of the entire vision, scintillating streaks or spots, or loss of half the vision periodically precede the headache, but there are no other symptoms of eye strain during or between the attacks.

Again, such a patient may have repeated nasal packs or a septum or sinus operation by a rhinologist, even though the history gives no suggestion of definite nasal or sinus symptoms and very often no physical evidence of such involvement.

If in addition to the frontal headache there are symptoms of nausea and vomiting, these patients may submit to the removal of the appendix or gall bladder by the general surgeon.

The gynecologist, if consulted regarding attacks of periodic headache, may consider these attacks as nerve reflex phenomena, indirectly caused by some pelvic pathology.

If associated symptoms such as aphasia, amnesia, vertigo and paresthesia are present, and they often are in the migraine patient, a neurologist or a neurosurgeon may come into the diagnostic picture.

A great many patients who have suffered with periodic vasomotor headache with various associated symptoms for years have had many types of treatment and operation, but only on the basis of a treatment diagnosis.

By *differential diagnosis*, I mean just what the term implies. A careful history taken by the physician himself is usually of much more importance than the physical examination. Really, to evaluate the patient's complaints and clarify them properly is a tedious but scientific procedure in the handling of headache patients. Many of the patients come to us with a preformed idea, or even conviction, as to the cause of their periodic headaches. In many instances this idea has developed gradually through the years because of various treatments or operations advised after only a treatment diagnosis. If various physical findings, X-ray and laboratory evidence are given undue importance especially without a proper history, the differential diagnosis is commonly confused rather than clarified. In order to make a proper diagnosis in a patient with periodic headache or facial pain of probable vasomotor origin, the examiner must first of all approach the problem as a physician, and secondarily as a specialist in any particular field. Many, many times the treatment diagnosis of the probable cause of periodic headache, with or without associated symptoms, depends mainly on the specialty of the physician whom the patient consults.

IMPORTANCE OF ASSOCIATED CORTICAL SYMPTOMS IN DIFFERENTIAL DIAGNOSIS.

Scintillating scotomas (dancing, zigzag lines or spots before the eyes), *hemianopsia* (loss of vision in one field), and blurring of the entire visual field, are typical prodromal symptoms in many patients with migraine, and certainly repetition periodically of such symptoms with periods of normal visual function must suggest that the pain in the head and other associated symptoms which follow must have a vasomotor basis. In many migraine patients, one of these visual phenomena always precedes the pain and the other symptoms and usually persists for 15 or 20 minutes. Some patients, as they grow older, may experience only the visual phenomena without any headache or other associated symptoms.

Paresthesias (numbness in hand, arm, lips or tongue) commonly appear with the visual symptoms or soon after they have developed. If this numbness appears on the left side, the headache will usually be present on the right side, which, of course, suggests the cortical involvement of the sensory area on the right side of the brain. Such symptoms, because they come first and are of short duration, are elicited only by a careful history.

Nausea and *vomiting* are commonly present and usually are most troublesome when the headache is most severe. These symptoms are much more common and prominent in the child and the young adult than in the older patient. In fact, it is almost axiomatic that their severity varies in inverse proportion to the age of the patient. Real gastric or intestinal conditions rarely cause headache of any severity; but many severe gastric and intestinal symptoms follow or are associated with headache of vasomotor origin and are entirely absent between the periodic attacks.

Vertigo with pallor, cold perspiration, nausea and vomiting often appear as prodromal symptoms in an attack of migraine. What is of more importance, however, is that this symptom complex replaces the entire migraine syndrome in many patients for months or years. Such attacks are typical of Ménière's syndrome except that tinnitus and loss of hearing are not present. These attacks, because of their short duration and lack of after-effects, though they are common,

are seldom diagnosed. Rarely does an otolaryngologist see such patients at the time of their attacks, and rarely does the general physician, who usually does see them, recognize the real nature of the disturbance. The diagnosis is usually heart trouble or acute indigestion, because the gastrointestinal symptoms and the appearance of the patient are dramatic, and little if any attention is paid to the causative vertigo. As a rule, the general physician need not give much thought to the actual nature of the disturbance or to call the otologist in consultation, because the attacks are of short duration and the patient is up and as well as ever in a few minutes, an hour or a day. The patient usually recovers quickly, regardless of and usually despite the treatment advised.

Aphasia and *amnesia* of short duration are fairly common in typical cases of migraine. These symptoms, however, are rarely mentioned voluntarily by the patient because of the secret fear that they indicate a real mental disturbance. Many other patients will not mention these complications in the ordinary history because previously, when they did go over their problems with some physician, they were told almost flippantly that they were "just nervous." Fortunately, these symptoms are transient and are present only when the headache or some other associated symptoms are demanding most of the attention.

Symptoms referable to the *motor area of the brain* are mentioned simply to emphasize that apparently they rarely, if ever, appear during attacks of vasomotor origin. I have yet to see a patient with epileptic seizures during or after a vasomotor outburst. Naturally, it would be possible for epilepsy and migraine to appear in the same person as a coincidence, but in my experience this association of the two conditions has not occurred.

Comment: The associated symptoms mentioned, when they occur periodically, do play a great part in helping to make a differential diagnosis of the probable type of periodic headache or neuralgia in any given patient. All of these symptoms may appear in a periodic routine in some patients, some of them in other patients, and none in many. It is important to emphasize that probably no more than 25 per cent of patients suffering from periodic vasomotor headache have any of these associated symptoms. If, however, one or more

of them appear in sequence during many periodic attacks of headache or neuralgia, with intervening periods of good health, the diagnosis is practically made, regardless of, and often in spite of, physical findings or other symptoms which have no bearing on the proper differential diagnosis. Physical findings, especially Roentgen evidence, may easily confuse the issue because of their incidental presence in a region anatomically closely related to the location of the pain. And pain, we must always remember, is the main point of argument in any debate involving the patient, nature, and the physician. No one of the individual associated symptoms is ever as typical as the periodicity of a certain number of them over long periods of time and the regularity of the progression of these symptoms during each attack in a patient whose personal and family history suggests possibilities of allergy or migraine. Certainly, in many family histories, hay fever, asthma, urticaria and eczema may be interchangeable with migraine or some type of allergic or vasomotor headache.

Pain in the head or face, periodic in nature and accompanied by such cortical symptoms as have been mentioned, occurring in various combinations can have no other basis than temporary tissue or fluid changes in the brain, meninges, ganglions or nerve sheaths. Real pathologic changes in the nose, sinuses, eyes, teeth, brain or any other part of the head cannot be responsible for such systematic reproduction of similar explosive phenomena over relatively long periods of time with intervening periods of good health.

MIGRAINE IN CHILDREN.

Migraine, or some type of vasomotor or allergic headache with associated symptoms, is much more common in children than is generally realized and deserves special mention in this paper. Little attention has been given to the subject in the textbooks on pediatrics or even in the current literature of this specialty.

At the age of 4 or 5 years, or earlier, the headache itself is often a minor symptom of the periodic attacks, which are characterized by languor, loss of appetite, fever, abdominal discomfort and vomiting. Elevation of temperature, even to high levels, is common, and it may reach 105° or 106° F. within a few hours after the beginning of the attack and then

return to normal by the next day or sooner. The gastric disturbance in the young child is most marked, and nothing may be said either by the patient or by the parents concerning the headache or other transient prodromal symptoms. Many children have associated symptoms, such as scotomas, general blurred vision, aphasia and paresthesias at the beginning of the attacks, but the existence of such prodromal symptoms may be elicited only by careful attention to the history.

CLASSIFICATION.

As I have said previously in this paper and in several others, history is of much more importance than physical examinations of all types as an aid to a proper differential diagnosis as to the probable type of periodic headache or facial neuralgia, especially in the ambulatory or office patient who usually has good health between the periodic attacks. It seems to me that a proper classification of the usual types of headaches and neuralgia is of great importance in enabling one to take a proper and useful history. Classifications of headache and neuralgia as they usually appear in the literature have seemed to me to be confusing and impractical for the use of the practitioner of medicine, regardless of his specialty, who hopes to apply what he reads to what he does.

I am submitting a classification at this time which has developed gradually through the years as a result of my personal experience as a victim of migraine and my experience with patients suffering with similar periodic headaches and neuralgias of probable vasomotor origin in the everyday private practice of ophthalmology and otolaryngology. Such a classification, I hope, will be helpful to anyone in our specialties who is definitely more interested in differential diagnosis than treatment diagnosis. Naturally, treatment is the goal in all of our efforts to make a differential diagnosis, but such treatment, to be proper, ethical and reasonably successful, must follow rather than precede proper diagnostic efforts.

HEADACHE AND FACIAL NEURALGIA – CLASSIFICATION FOR EVERYDAY PRACTICE.

Headache or *Neuralgia* as *Incidental Symptoms:*

Usually bed or hospital patients.

Other symptoms and physical signs more important.
Usually not periodic with intervals of good health.

Differential Diagnosis: Relative to main disease syndrome.

Headache or *Neuralgia* as *Main Symptoms:*

Usually ambulatory or office patients.
Other symptoms and physical signs less important.
Usually periodic with intervals of good health.

Differential Diagnosis: Periodic headache and neuralgia.

I. *Acquired Conditions:* Problem of *individual:*

1. *Nerve-end* Syndrome: local origin:

   a. Eye Type: ciliary and extraocular muscle strain.
   b. Nose and Sinus Types: pressure — inflammation — infection.
   c. Dental Types: caries — impactions — malocclusion.
   d. Neuralgia and Neuritis: idiopathic origin.

      1. Gasserian, sphenopalatine— geniculate ganglia.
      2. Vth, VIIth, IXth nerves.
      3. Vidian, great superficial petrosal.
      4. Myalgic or Indurative (neck syndrome).

2. *Reflex* Syndrome: general origin.

   a. Nerve routes from elsewhere in body.

3. *Vascular* Syndrome: general origin.

   a. Toxemia: gastrointestinal (histamine) — focal infection, etc.
   b. Drugs, chemicals, alcohol.
   c. Endocrine imbalance.

II. *Hereditary Conditions:* Problem of *Ancestry:*

1. *Vasomotor* Syndrome: Periodic transient tissue reactions in brain, meninges, ganglia and nerves.

   a. Vasomotor Headache: with or without associated symptoms.

   b. Migraine: cortical symptoms (hemianopsia, paresthesias, scotoma, nausea, vomiting, vertigo).

c. Allergic Headache: other evidence of allergy.

d. Myalgic or Indurative (neck syndrome).

e. Vasomotor Neuralgia and Neuritis.

1. Gasserian, sphenopalatine — geniculate ganglia.

2. Vth, VIIth, IXth nerves.

3. Vidian, great superficial petrosal nerves.

In the above classification, it will be noted that there are two main divisions; *viz.*, headache or neuralgia as incidental symptoms, and headache or neuralgia as main symptoms.

HEADACHE AND FACIAL NEURALGIA AS INCIDENTAL SYMPTOMS.

As a rule, when these symptoms are incidental, other symptoms and physical findings are more important and demand most attention. Usually, the patients are suffering from some acute illness which confines them in the home or in the hospital. The symptom complex then under consideration usually does not appear periodically with intervals of good health. Naturally, then, the main diagnostic effort is directed toward a probable disease which is self-limited by recovery or death. As far as this paper is concerned, nothing more will be said regarding the incidental appearance of headache or pain in the face as these symptoms appear in the course of disease syndromes, such as the acute infections, meningitis, encephalitis, brain abscess or tumor, kidney or circulatory involvements, etc.

HEADACHE AND FACIAL NEURALGIA AS MAIN SYMPTOMS.

At this time, and in everyday practice, my main interest is in this second group, in which headache or facial neuralgia are main symptoms. Patients under consideration are usually ambulatory or office patients and their attacks are periodic in nature, usually over long periods of time, with intervals of good health. Other symptoms and physical signs are of less importance in differential diagnosis, except as they are associated periodically with these main symptoms and disappear from the clinical picture between the attacks.

Differential diagnosis in the patients with periodic headache and neuralgia may be simplified at the outset if after a careful history one can be reasonably sure whether or not he is dealing with *acquired* conditions, which are the problem of the *individual*, or *hereditary* conditions, which are the problem of *ancestry*.

For the purpose of differential diagnosis, let us think of the headache or face pain secondary to acute nasal or sinus inflammation or infection, strain on ciliary or extraocular muscles, or dental pathology, as due to nerve-end irritation, mechanical or toxic in origin, but with the real pathology or source of irritation external to the brain and meninges. The actual pain, then, is reflex in nature.

On the other hand, let us think of the headache or face pain of the vasomotor type as due to changes in the volume of the brain substance, ganglion or nerve tissue secondary to transient local tissue reactions, or variations in the concentration of normal fluids. We can at least assume, though we cannot prove, that the tissue reactions in the brain which account for the various cortical symptoms in migraine are probably similar to the lesions of urticaria, which are so commonly seen in the skin.

In the differential diagnosis of the probable cause of periodic headache with possible associated symptoms, it is of utmost importance to keep this distinction between the hereditary and acquired types constantly in mind. In actual clinical experience, the periodic headaches or neuralgias of the acquired type such as result from eye strain, sinus infection, nasal obstruction or dental pathology never have any of the cortical symptoms such as scotoma, hemianopsias, paresthesias or aphasia, and rarely have nausea and vomiting, at least as an actual part of the headache or neuralgia syndrome. Certainly, in conditions of acquired origin there is no tendency for the various symptoms to exhibit any definite periodicity or tendency to progress routinely through similar phases during each exacerbation of the disability, with intervals of good health, which are characterized by lack of symptoms and physical signs.

All operations and many forms of treatment may act as "shock therapy" and give temporary relief from periodic vasomotor headache or any type of vasomotor disturbance, whether that disturbance be migraine, hay fever, asthma or some other syndrome with an hereditary basis.

Spontaneous relief, temporary or permanent, of periodic headache, or neuralgia, with or without associated symptoms of vasomotor or allergic origin, is more common than is similar relief from the combination of all treatments and operations in the otolaryngologic field and all other branches of

medical practice. Actually, just getting older accounts for the largest group of so-called "cures," regardless of whether or not there has been any treatment or even a diagnosis.

Patients suffering from migraine or some other form of hereditary vasomotor pain in the head or face with possible associated symptoms often should have nasal obstructions removed, sinuses drained, glasses prescribed, appendices and gall bladders removed, pelvic surgery done, and various treatments given, but only when there are good and sufficient reasons for doing these things, regardless of the vasomotor or allergic syndrome, and not because of it.

SUMMARY.

Migraine and similar familial allergic or vasomotor disturbances of the brain, meninges, ganglions, nerve sheaths and other bodily structures are very common and probably will continue to be very common as long as hereditary characteristics are perpetuated by intermarriage without thought being given to eugenic selection. Reduction in the numbers of prospective victims is probably more a social than a medical problem. It is to be hoped that in the special fields of allergy, endocrinology, or elsewhere, constant scientific effort will find better methods to alleviate the symptoms more commonly over longer periods of time in a greater percentage of the patients who are predestined by heredity to suffer with these periodic physiologic upsets in the brain or other tissues which are otherwise normal.

In the special fields of ophthalmology and otolaryngology, if a proper differential diagnosis is made, there is a wonderful opportunity to rule out many things which are not causing the trouble, and thereby save many patients from unnecessary and ill-advised treatments and operations.

Because the subject of the differential diagnosis of headache is too big to be covered in any one paper, I am listing other papers in which many phases of the subject are discussed more completely.

REFERENCES.

1. GITTINS, T. ROY: Differential Diagnosis in Headache of Periodic Type. *Ann. Otolaryngol., Rhinol. and Laryngol.*, Vol. 42, June, 1933.

2. GITTINS, T. ROY: Headache: Differential Diagnosis in Periodic Types. *Jour. Iowa Med. Soc.*, Sept., 1935.

3. GITTINS, T. ROY: Headache from Pathologic Changes in the Nose or Other Causes. *Arch. Otolaryngol.*, 30:589-600, Oct., 1939.

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A CONSIDERATION OF ATROPHIC RHINITIS AND ITS TREATMENT BY THE USE OF ESTROGENIC SUBSTANCE.*

DR. ARTHUR J. WAGERS, Philadelphia.

Before referring to our personal experience in the use of estrogenic substances in the treatment of atrophic rhinitis, let us consider briefly the background — the train of observations and study leading up to the employment of this particular form of therapy.

That there exists a connection between the sexual impulse and the nasal mucous membrane in man has been observed all the way from ancient times down to the present. There is no need at this time to go into detail regarding this biological phenomenon familiar to most of you, but for those especially interested I would recommend reading the admirable historical review by Dr. John Noland Mackenzie entitled, "The Physiological and Pathological Relations Between the Nose and the Sexual Apparatus of Man," and published in *The Johns Hopkins Bulletin* of Jan. 28, 1898. After referring to the observations of writers from the time of Hippocrates down, Dr. Mackenzie's paper concludes in these words:

"These observations, therefore, encourage the belief, if they do not establish the fact, that the natural stimulation of the reproductive apparatus, as in coitus, menstruation, etc., when carried beyond its normal physiological limits, or pathological states of the sexual apparatus in certain diseased conditions, or as the result of their overstimulation from venereal excesses, masturbation, etc., are often the predisposing and occasionally the exciting causes of nasal congestion and inflammation and perversion of the sense of olfaction. Whether this occurs through reflex action, pure and simple, or as a sequel of an excitation in which several or all of the erectile tissues of the body participate, the starting point of the disease is, in all probability, the repeated

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stimulation and congestion of the turbinated erectile tissue of the nose. It is highly probable that this erectile area or organ, so sensitive to reflex-producing impressions, is the correlative of certain vascular areas in the reproductive tract, and that the phenomena observed may, therefore, be explained by the doctrine of what we may call, for want of a better name, reflex, correlated action. The study of the relations between the nose and the sexual apparatus opens up a new field of research, of pleasing landscape and boundless horizon, which bids to its exploration not only the physiologist and pathologist but also the biologist. Above all, it brings us face to face with a serious problem of life and an interesting enigma, whose significance it will be the task of the future to divine."

Nearly 40 years later, Mortimer[1] and his associates in Montreal began a series of scientific investigations looking to a solution of this "interesting enigma."

From their study of the effects produced by estrogens on the mucous membrane of monkeys, they observed that the changes attributed to these substances were not only apparent in the sex-skin of the perineum, skin of the face and in the nipple, but also in the turbinal mucosa of the normal animal.

They summarize their observations as follows:

1. Crystalline estrone and estriol and emmenin cause redness and swelling similar to and, for the most part, synchronous with changes already known to occur in sex-skin areas.

2. This effect can be obtained with estrone and estriol in castrated animals.

3. It can be produced in males as well as females, in immature as well as mature animals. Emmenin by mouth enhances what we describe as the normal premenstrual peak — makes more evident the nasal evidence of sexual cyclicity.

4. Such peaks acquire clear significance when the average time between their occurrence is observed. In seven animals so treated, the average interpeak interval was found to be 26½ days.

They also made observations on pregnant women and found redness and swelling of the turbinates as early as the second month in a few cases, and found such change to be definitely apparent from the fourth to the ninth month — the change increasing as the pregnancy advanced. From all this they reached the conclusion "that the hormones of the ovary exercise a specific, physiological influence upon the mucous membrane of the conchae, both during the menstrual cycle in the monkey and in pregnancy in the human subject." Their histological study shows, experimentally, that in the monkey the nature of this change is a hyperemia followed by a perivascular edema in the submucosa. When this is excessive it leads to cellular infiltration and permanent enlargement of the conchae may result. Such a change, of course, should prove of distinct benefit in the condition designated "atrophic rhinitis."

It was from consideration of these findings that we undertook to observe the effect of treatment by estrogenic substance on such cases as presented themselves at the nose and throat clinic of Jefferson Hospital. During a period of 15 months we were able to observe and treat 26 cases. The number, while small, is perhaps sufficient to provide a reasonable indication of what may be expected from the treatment given.

Diagnosis: In atrophic rhinitis we are dealing with a condition essentially chronic. In fact, there appears to be no evidence to show that it ever passed through an acute stage. So far, I have never inspected the normal nose of any individual at a given time, and again at a later period, and found the nares presenting the typical appearance of atrophic rhinitis. The condition is first seen fully developed or not seen at all. Patients themselves usually give only vague replies to questions asked concerning duration — "four or five years, maybe longer" — "since I was about 10 years old" — "all my life," are characteristic replies. In selecting our cases we have done so on the basis of the history given and the objective findings — the latter being more important than the former. In making examination of these cases we must not lose sight of the fact that the judgment of individual examiners is likely to vary — what appears to one to be a considerable mass of crusty material seen in the naris, to another might appear to be a comparatively small amount. Judgment will

also vary as to the spatial aspect of the naris or in the observance of ozena. In other words, there are no definitely fixed standards by which we can measure our findings either before or after treatment. Ozena was not observed in all our cases and certainly it was much more pronounced in some than in others.

Patients do not always complain of the same group of symptoms. In most instances their discomfort is attributed to dryness or crusting in the nose, some complain of anosmia, and others are disturbed by headache or "bad odor on the breath"; however, this latter discomfort is not likely to be noted so much by the patient as by his friends or family.

Our own diagnosis was finally based on the wide open naris, particularly in the region of the lower turbinate. This structure appears much smaller than the average; in fact, many of them appear to be rudimentary. Clinically, there were none in this group suffering from active sinusitis, although Roentgenographic studies showed mucous membrane thickening in about one-third of the cases.

Treatment: Treatment was uniform. Patients visited the clinic once weekly, at which time the nasal passages were thoroughly cleansed and amniotin* (10,000 units) was applied to the surface of the nasal mucous membrane. The patient was given a supply of this estrogenic substance to be used at home after irrigating each nasal passage with two quarts of warm normal saline solution, to which was added 4 gm. of sodium bicarbonate. This solution was allowed to flow through the nose slowly and under minimum pressure. This cleansing and the use of the estrogenic substance from an atomizer was done three times daily over a period varying from eight to 12 weeks, when these home treatments were done less frequently — once daily for two weeks; then, if the nasal condition appeared to warrant doing so, the estrogen was discontinued, both at the clinic and at home; however, the patient continued one daily irrigation during the remainder of time he was under observation.

Of the 26 patients treated, 11 were males and 15 were females. Ages ranged from 6 to 72 years, the average being 31 years. There was no evidence of sinusitis in 21 cases,

*Amniotin used in the treatment of this series was generously supplied by E. R. Squibb and Sons.

while five others presented sinus involvement in varying degrees of chronicity. Ozena was present in 13, or one-half the cases.

Result of Treatment: The word "cure" has not been applied to any case, because in no instance have I seen anatomical structure or physiological function fully restored. I have chosen rather to use the word "improved," dividing the treated patients into three groups according to the degree of improvement noted. This, of course, is an arbitrary grouping and is based on the appearance of the nares and the patient's own estimate of relief experienced. In those cases showing *1.* marked improvement, the nares remained quite free from crusting and ozena, when the latter had been present at the beginning of treatment, and the patient expressed himself as decidedly more comfortable. There were but six patients in this group. The next group, 12 in number showed *2.* moderate improvement. They were still troubled by a certain amount of crusting from time to time, with occasional recurrence of ozena, and the feeling of general relief was not so marked as in the previous group. In the remaining eight cases there was but *3.* slight or no improvement. It should be said of this group that they were somewhat irregular in attendance at the clinic and more or less neglectful in their home treatment.

Thus we see that, taking the first two groups together, there were 18 cases, or 69 per cent of the whole number, in the series who showed some degree of clinical improvement, while in the remaining 31 per cent the improvement was too slight to be designated as even moderate.

Observations on conditions found in certain individual patients may be of interest. For instance, two patients complained of severe and rather constant headache not due to active sinusitis. One of these was a young woman, age 20 years, who presented the typical wide open nares with considerable crusting. Anosmia had been noted during the past six years. Ozena was present in slight degree. Her chief complaint was severe pain over the nasion. She showed marked improvement after about six weeks of treatment, the nose being free from crusting, head pain entirely relieved, and she was generally much more comfortable. The other case was also that of a young woman, age 34 years, who had

a past history of sinusitis with radical antrum operation on one side. She was free from sinus activity at the time of beginning treatment with estrogenic substance. She also complained of severe pain over the nasion, which was relieved by treatment for the atrophic condition. The latter, however, showed only moderate improvement, there still being recurrences of crusting and a certain amount of ozena at times. In both these patients inquiry was made as to menstrual history and the first case was found to have been normal as to time of appearance of menses and regularity. In the second case, menstruation had begun at the age of 16 years and for some time had been regular and lasting one or two days. During the past four years, however, there had been some irregularity, the menses being spaced at six- to eight-week intervals but still lasting only one or two days.

Other cases presented menstrual irregularity and also growth irregularities suggesting dyspituitarism. Perhaps the most interesting of these was a young girl, age 14 years, when first seen. She had greatly widened nares filled with very foul-smelling crusts — the typical atrophic rhinitis at its worst. This girl was small for her age, appearing to be not more than 10 or 11 years. In December, 1939, when she was 15 years and 8 months of age, she was four inches under height and 30 pounds under weight. Her first menses had appeared one month before this time and had lasted four days. After 10 months' estrogenic treatment this patient showed only a moderate degree of improvement, there still being constant tendency to crust formation and the ozena, while not nearly so marked as in the beginning, was still present. While this patient's development suggests hypopituitarism, there was the case of a boy, age 6 years, who appeared to be at least two years older, suggesting hyperpituitarism. He presented the typical nasal atrophic condition and was one of the patients showing marked improvement under treatment.

The patient showing the greatest menstrual irregularity was a woman, age 24 years. Her periods had always recurred at intervals of three to four months and had always lasted seven days. As for the nasal condition, she stated that she had suffered from heavy nasal crusting and marked ozena from early childhood. The degree of improvement in this case was classed as only moderate.

Study of these cases, while too few in number to justify conclusions, does further suggest the possibility of early endocrine dysfunction, involving especially the ovaries and the pituitary gland. Research being carried out in various quarters at the moment will, it is hoped, make clear just what lies behind this condition we designate "atrophic rhinitis."

In the light of our present knowledge, there are questions which stand out and require answers, for which time and observation will be needed before they can be answered satisfactorily. One of the very important questions has to do with the permanency of results obtained by the use of estrogenic substance in treatment. Let us assume for the moment that we have a patient presenting the typical picture of atrophic rhinitis with widened nares filled with foul-smelling crusts. We assume that this is probably the result of hormone deficiency, at least that this is a principal factor, and we attempt to supply this deficiency by the surface application of an estrogenic substance to the nasal mucous membrane. Let us further assume that this treatment does bring about highly satisfactory results from the clinical standpoint; we are now confronted by the question, "Will these results show any degree of permanency after we discontinue treatment?" Time will answer the question for our series, but for the present I am doubtful.

REFERENCE.

1. Mortimer, H.; Wright, P.; Bachman, C., and Collip, J. B.: The Effects of Administration of Estrogenic Hormones on the Nasal Mucosa of the Monkey (Macaca Mulatta). *Proc. Soc. Exper. Biol. and Med.*, 34:535, 1936.

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HORNER'S SYNDROME WITH CHRONIC PURULENT OTITIS MEDIA. DEMONSTRATION OF A DIAGNOSTIC TEST.*

DR. LOUIS HUBERT, New York.

In the German and French literature there are recorded a few observations on involvement of the sympathetic nervous system of the eye resulting in a Horner's syndrome, from chronic lesions of the tympanic cavity. As far as I know, no cases have been reported in the American literature; however, there is abundant experimental evidence in both the foreign and American literature that destruction of the tympanic cavity in animals will produce this syndrome. The physiological experiments of Metzner and Wolflin[1] in 1914 on the rabbit, of Kleyn and Soccin[2] in 1915, of Jarcho and Root[3] in 1940 on the cat agree with the above clinical observations.

In the anatomical laboratory of Lille, France, Piquet and Coulouma[4] investigated in men the anatomical distribution of the nerve fibres at the level of the promontory and the relationship of the sympathetic plexus in the vertical portion of the carotid canal. Their studies show that often several filaments from the carotid sympathetic plexus detach themselves and penetrate the middle ear cavity while traversing the anterior wall. They travel to the anterior part of the promontory underneath the mucous membrane and sometimes in the depth of the bone of the anterior wall to the petrosa. This explains why Jarcho and Root[3] observed in the physiological laboratory of Columbia University sympathetic ocular phenomena following labyrinthectomy on cats. The sympathetic filaments coming from the carotid plexus and traversing the tympanic cavity rejoin this plexus and finally reach the dilator muscle of the pupil by accompanying branches of the long ciliary nerves. The majority and sometimes all the sympathetic ocular fibres continue along the carotid plexus without entering the middle ear cavity. This would

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explain the rarity of a Horner's syndrome in middle ear disease of men.

REPORT OF A CASE.

G. S., female, age 37 years, came to the Manhattan Eye, Ear and Throat Hospital, clinic of Dr. Francis W. White, on March 27, 1941, complaining of pain in the right half of the head and discharge from the right ear. Ten years ago she suffered from a right purulent otitis media and after two years of conservative treatment a radical mastoidectomy was performed in another hospital. Within a short time after this operation, a drooping of the right upper lid was noted. Examination also revealed a constriction of the right pupil, a narrowing of the palpebral fissure and an apparent enophthalmos of the right eye. These signs constitute a Horner's syndrome. Examination of the right ear showed a partial atresia of the right auditory canal. X-ray of the right mastoid suggested the presence of a cholesteatoma. A secondary mastoidectomy was indicated, which the patient was not willing to undergo at present. Blood and urine tests were negative and there were no other neurologic findings. There were no glandular enlargements and no suspicion of a growth in the nasopharynx. The Horner syndrome could not be explained on a basis other than the chronic otitis.

DIAGNOSTIC TEST.

The drugs used were a 4 per cent solution of cocaine and a 1:1,000 solution of epinephrine. Instillation of a 4 per cent solution of cocaine into the conjunctival sac of a normal person will cause a dilatation of the pupil. In the presence of a Horner's syndrome such a dilatation will not occur.

It is generally believed among ophthalmologists that the instillation of epinephrine hydrochloride into the conjunctival sacs of normal persons also causes a dilatation of the pupil. This is contrary to the findings of Loewi,[5] one of the winners of the Nobel Prize in medicine and physiology in 1936. He has shown that when epinephrine hydrochloride is instilled into the conjunctival sacs no mydriasis will ensue; however, if the sympathetic nervous system is rendered hyperirritable, as may occur in persons with exophthalmic goitre or diabetes, dilatations of the pupils will become apparent. In applying this test to many hundreds of patients in the Manhattan Eye, Ear and Throat Hospital during the last 25 years, I have found the observations of Loewi to be correct; moreover, I have noticed other conditions that may produce unilateral hyperirritability of the sympathetic nervous system of the eye and unilateral dilatation of the pupils after the instillation of epinephrine hydrochloride. This phenomenon has a certain diagnostic significance which I have dis-

cussed in a paper published in 1937.[6] In order to understand the effect of epinephrine on the pupil, as well as on the smooth muscle fibres of the lids and Mueller's orbital muscle, it is well to remember a fact long known to physiologists; namely, that denervated smooth muscles become sensitive to the circulating epinephrine in the blood and, of course, will become still more sensitive when this substance is directly applied by conjunctival instillation. If the dilator muscle of the pupils and the other bundles of smooth muscles of the eye are cut off from their nerve connection with the superior cervical ganglion (*i.e.*, if the lesion is in the ganglion or its postganglionic fibres), epinephrine hydrochloride instilled into the conjunctival sac will act directly on the denervated muscle fibres and cause not only dilatation of the pupil but elevation of the lid and an apparent slight protrusion of the eyeball. This reaction has been obtained in the case reported in this article, showing that the lesion is in the postganglionic fibres.

In the clinics of the Manhattan Eye, Ear and Throat Hospital I often find patients with paralysis of the cervical sympathetic system resulting in Horner's syndrome. The lesion causing this condition may be in the superior cervical ganglion, the preganglionic and postganglionic fibres or the central nervous system. Usually it is easy to locate the lesion causing the syndrome. Occasionally the instillation of epinephrine hydrochloride into the conjunctival sac may be the only means of localizing it. It will tell one with a reasonable degree of certainty whether the lesion is *1.* in the superior cervical ganglion and its postganglionic fibres, or *2.* in the preganglionic fibres and the central nervous system. In the first instance there results denervation of the smooth muscles of the eye supplied by the sympathetic nervous system and hypersensitiveness to epinephrine. In the second instance no denervation and no hypersensitiveness follow.

The epinephrine test is especially useful in the early diagnosis of malignant tumors of the nasopharynx. The early symptoms of such tumors are usually vague and uncharacteristic. Because of the failure to identify a small lesion in the region of the Eustachian tube, the diagnosis is often overlooked. The close relation of the tumor to the base of the brain and its tendency to invade cause lesions of the cranial nerves, especially those of the eye. In the presence of a Horn-

er's syndrome, which cannot be accounted for by a neurological examination, the epinephrine test may be the only deciding factor in the diagnosis. At any rate it will give additional information when the objective examination is not conclusive. Repeated biopsies of any suspicious appearing tissue from the nasopharynx in the region of the Eustachian tube may reveal the primary lesion. While the prognosis in these tumors is grave, Roentgentherapy, if given early enough, may lead to recovery. The value of this test, therefore, in arriving at an early diagnosis, cannot be overestimated. A dilatation of the pupil after the instillation of epinephrine indicates that the postganglionic fibres are involved. That should make us strongly suspect a malignancy of the nasopharynx, if other causes are excluded. On several occasions this test has aided me in arriving at an early diagnosis. In performing this test, one must be patient to wait for the reaction to occur. It may take an hour or longer, depending upon the color of the iris. The darker the color of the iris the longer it takes to get the reaction.

REFERENCES.

1. Metzner and Wolflin: Klinische und experimentelle Untersuchungen ueber Halssympathicus Laehmung. *Arch. f. Ophth.*, 84:308, 1914.

2. DeKleyn and Soccin: *Arch. f. d. ges. Physiol.*, 1915.

3. Jarcho, L. W., and Root, W. S.: The Relation of Labyrinthectomy to Sensitization of the Nictitating Membrane in the Cat. *Am. Jour. Physiol.*, 128:526, 1940.

4. Piquet, J., and Coulouma, P.: Les signes oculo-sympathiques dans les otites et leurs raisons anatomiques. *Ann. d'oto-laryngol.*, p. 1003, Sept., 1935.

5. Loewi, Otto: Ueber eine neue Funktion des Pankreas und ihre Beziehung zum Diabetes mellitus. *Arch. f. exper. Path. u. Pharmakol.*, 59:83, 1908.

6. Hubert, Louis: Diagnostic Significance of Epinephrine Instilled into Conjunctival Sacs. *Arch Ophth.*, 17:1076, 1937.

SUPPURATIVE ETHMOIDITIS WITH SPECIAL REFERENCE TO ORBITAL ABSCESS AND TREATMENT.

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No complication of ethmoiditis is more serious to life and to structure as is rupture of an ethmoid cell associated with abscess in the orbital cavity. Meningitis caused by extension through the venous channels, as well as brain abscess and cavernous sinus thrombosis can be caused by diseased ethmoids, particularly by an acute exacerbation of a chronic pathology.

Rupture of an ethmoidal abscess into the orbit, although not uncommon, is still a most dangerous complication. Early recognition of the clinical picture and proper surgical procedure instituted may not only save the patient's life, but early interference may avoid the most dreaded complications, namely, blindness of the eye on the affected side, and danger to life.

It is true that dehiscences in the bony structure of the ethmoid labyrinth may occur, but necrosis due to repeated infection in a chronic sinusitis can and will break down the thin wall of the lamina papyracea.

Anatomy: The ethmoid cells develop early and are clearly present at birth. The ethmoid bone consists of a perpendicular plate, a horizontal plate and two bodies called the ethmoid labyrinths. The perpendicular plate is attached to the central part of the caudal surface of the cribriform plate and extends downward between the two labyrinths.

It articulates in front with the triangular cartilage of the septum and posteriorly with the vomer. The horizontal plate, known as the cribriform plate, is perforated for the olfactory nerves and is the only portion of the ethmoid directly in contact with the dura mater. The ethmoidal labyrinth, or lateral masses, comprise the greater part of the ethmoid. They are symmetrically placed, one on each side of the nasal cavities.

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The labyrinth has three surfaces, the superior, lateral and medial. The lateral surface is also known as the lamina papyracea. This portion of the ethmoid consists of extremely thin bone and forms the medial wall of the orbit. It is a somewhat rectangular, smooth area of bone, measuring 2.5 cm. in length and 1.5 cm. in width.

The ethmoid labyrinth contains the cells. It is about 1.4 cm. in width. Sometimes the cells lie in layers, an upper and a lower. Irregularity in the position of the cells is not uncommon. The cells are divided into an anterior group and a posterior group. The former empties into the middle meatus

Fig. 1. F. W., showing the swelling over the left eye.

with the antrum and frontal sinuses, while the posterior group opens into the superior meatus, together with the sphenoid.

Treatment: No condition in the field of otolaryngology requires more urgent attention than infection of the orbital space. It is true, surgical interference need not be depended upon at all times. Experience and proper judgment will tell us just what procedure to follow. It has been the experience of most otolaryngologists, particularly those associated with active hospital services, that surgery such as external incision and drainage over the area of pointing should be used in adults, and that this procedure be followed, after a few days when the acute condition had quieted, by complete exenteraation of the ethmoid labyrinth, endonasally.

REPORT OF CASES.

F. W., female, age 42 years, was admitted to the hospital, July 13, 1938. Ten days before admission, she caught "cold" in the head. Her left eye began to "swell." She had some pain in the region of the eyeball, which was aggravated by pressure. Her nose had been "running" only a little more

Fig. 2. F. W., X-ray of paranasal sinus shows involvement of the ethmoids, with a breaking through into the orbit.

Fig. 3. J. M., marked swelling over right upper eyelid and region of the inner canthus. Note narrowing of palpebral fissure.

than usual. She had no previous similar attack. There was no history of trauma and her temperature had not been taken at home.

On admission, she appeared acutely ill. Over the region of the left eye, particularly behind the upper lid and at the inner

canthus, there was seen a large mass, tender to touch, and soft. The upper eyelid moved with difficulty and caused great pain. The nasal septum showed an anterior perforation.

X-ray examination report showed the frontal sinuses as symmetrical, fairly well developed but cloudy. The left ethmoids were cloudy. The left antrum was cloudy.

The temperature was 100°, pulse 90, and respiration 25.

Right Left

Fig. 4. J. M., cloudiness of the right frontal, both right and left ethmoids and right antrum.

Blood Wassermann was negative. Urine was negative.

On July 14, 1938, under local anesthesia, the orbital abscess cavity was opened by incision in the upper eyelid at the point of greatest fluctuation. The cavity was packed with iodoform gauze. The local condition continued to improve. On the nineteenth day of July, under local anesthesia, I performed a Mosher technique endonasal left ethmoidectomy. On the twenty-eighth, the patient was discharged cured.

J. M., age 10 years, was admitted to the hospital on April 20, 1938, complaining of a swelling and redness of the right eyelids. His previous history is irrelevant. His present illness began one week previous to admission, with a sore throat

and some temperature (exact degree not known to patient). Two days after the onset he noticed the local condition over the right eye. It was painful to touch, and an attempt to open the eyelids caused pain and discomfort. This condition progressed until the right eye was completely closed. His sore throat by this time was better.

On admission the local condition, as shown in the picture, was marked chemosis of the conjunctiva of the right eye, swelling of the eyelids, and marked tenderness over the region of the inner canthus.

His nasal mucous membrane was swollen, inflamed and covered with a purulent exudate, especially in the region of the middle meatus and middle turbinate of the right side. Erosion of the septal mucous membrane on that side showed evidence of a recent epistaxis. The ears were negative. Examination of the eye showed no involvement of the iris, cornea or optic stalk. *There was no muscular impairment.* The regime instituted in this case was the application of wet boric acid dressings over the swollen eyelids, and intranasal instillation of 1-10,000 adrenalin solution, both in the form of spray and pack, followed by gentle suction.

The patient's temperature ranged between 98.6° to 103.8° (highest occurred on third day after admission).

X-ray examination showed cloudiness of the right frontal. Both anterior and posterior ethmoids were markedly cloudy. The right antrum was cloudy. There was some involvement of the floor of the left antrum. The right turbinates were hypertrophied and the septum deviated to the right.

The local condition continued to improve; the patient was discharged, May 12, 1938, entirely well.

COMMENT.

1. Two cases of ethmoiditis are reported, both with orbital infections.

2. Modus operandi in each are described.

3. Importance of early surgical interference in cases where pus has already developed.

BIBLIOGRAPHY.

BARNHILL, J. F.: Surgical Anatomy of the Head and Neck, 1937.
JACKSON and COATES: The Nose, Throat and Ear, 1929.

1749 Grand Concourse.

NAROMETRY. TESTING NASAL RESPIRATION.

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WHAT CONSTITUTES NORMAL NASAL RESPIRATION?

"The normal respiratory function of the nose with its pathologic deviations has not received the consideration it deserves by virtue of its great importance in the maintenance of good health and its influence upon the growth and the normal development of the young. While much good work has been done and much has been written on the gross and microscopic anatomy and pathology of the nasal structures and its accessory sinuses, comparatively little has been done and written on the subject of the physiology of nasal respiration."[1]

The difficulty in answering the foregoing question arises from two basic factors; namely, *1.* we must depend largely on subjective data, the history of the case and the patient's statements concerning his nasal respiratory capacity; *2.* the lack of reliable objective data, instruments of precision and tests to enable the examiner to establish a norm or standard of normal nasal breathing and to determine accurately any deviation from it.

While in the practice of otology and ophthalmology, standards for normality of audition and vision have been established and any degree of abnormality of these functions can be determined with exactness by means of precise instruments and tests, the same does not obtain in the field of rhinology where a criterion of normal respiration is lacking and functional tests are vague and unsatisfactory.

(A) Subjective Data:

1. History: As a rule the patient is rather vague about the onset of his respiratory difficulties, unless they are of an acute nature, such as an acute rhinitis, an allergic condition or an injury to the nasal structures of sufficient severity to cause an appreciable encroachment upon the air passages of

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the nose. On the other hand, where the process is gradual and slow in developing, the patient may not experience any respiratory deficiency for many years, or this inadequacy may be discovered by the routine examination of the physician and brought to his (the patient's) attention.

2. Present Status: While, in the final analysis, the patient's experience in the matter of his nasal respiratory capacity is of prime importance in arriving at a proper diagnosis of the case, yet we cannot at all times feel assured of the correctness of it unless we possess the means to compare and check his statements with our own objective findings. It may be to the patient's advantage to exaggerate his respiratory difficulties in case of an injury to the nose resulting from an accident where compensation is involved; or the patient may be a highly neurotic individual, in which case the slightest discomfort may be played up to tragic proportions. On the contrary, bilateral nasal respiratory inadequacy of a mild degree of many years' standing may be found in many patients without their being aware of it; or a unilateral obstruction of a marked degree, which almost occludes one nostril, may coexist with a perfectly functioning nostril on the opposite side without the patient's awareness of the condition. It is only when the normal nostril become inadequate, due to an acute coryza or some other obstructive condition creating a sense of air hunger, that the patient notices the incapacity of the opposite nostril.

(B) Objective Data:

1. Structural Examination — Anterior and Posterior Rhinoscopy: Direct and indirect inspection of the nasal structures is of the greatest importance in the rhinoscopic examination of the patient. The data furnished by a thorough and minute examination of the nasal passages is of inestimable value in the diagnosis of nasal obstructions and of nasal function. While in the main it is true that the normal or abnormal function of an organ depends in a great measure upon its anatomic structure, yet it is a matter of great difficulty to judge the function of an organ by its structural appearance, whether normal or abnormal, for the reason that alterations in the structure of a part do not keep pace with alterations in its function. By analogy the ear may be

considered. The visible pathologic changes in an ear under examination may be quite marked, such as complete or partial destruction of the eardrum, with other marked changes in the tympanum, and yet the function of the ear may be comparatively little impaired; on the other hand, we may encounter comparatively slight anatomic changes in the middle ear with a marked degree of conductive deafness. In a similar manner, we find on nasal inspection deviations from the normal which would lead us to assume great interference with the function of breathing, yet the patient may state that his breathing is quite normal or very slightly disturbed. On the contrary, in cases of slight nasal pathology or in cases of slight structural deviations the nasal respiratory function may be greatly hampered. The reason for these discrepancies between structure and function, especially in rhinologic conditions, lies in the fact that it will require much encroachment on the nasal air passages for the patient to experience any air hunger. Again, an apparently marked obstruction may not cause as great an alteration or interference with function as a minor one which lies directly in the path of the air currents; furthermore, in many cases of nasal respiratory difficulty the cause will be found rather extranasally than intranasally; *i.e.*, in narrowed nasal passages, in slightly collapsed alae nasi, or in a slightly downward prolongation of the same than in an obstruction within the nose.

2. *Functional Tests: (a) Auditory:* This consists in listening attentively to the patient's breath sounds during quiet and forced respiration as the air currents circulate through nasal passages during inspiration and expiration. Much valuable information can be gleaned from this procedure. In applying this method, each nostril is tested separately by closing the opposite one with the ball of the thumb so that no air can come through it and instructing the patient to breathe through the open nostril exclusively. Normal breath sounds are quiet and regular, whereas in any obstruction to the inspiratory or expiratory air currents the breath sounds will be labored, exaggerated or become barely audible, depending upon the amount or location of the obstruction. If the patient has one normally functioning nostril and a greatly obstructed one on the opposite side and the patient is instructed to breathe deeply through the defective nostril while the normal one is occluded with the ball of the thumb,

a loud popping sound is distinctly heard on the quick withdrawal of the thumb from the occluded nostril, due to the inrush of air into the free side.

(b) Tactile: This is divided into a positive and a negative phase. The positive phase, as practiced by otologists, consists in closing one of the patient's nostrils and instructing him to exhale vigorously while the examiner holds the dorsal surface of either hand near the patient's open nostril and feels the impact of the air current striking against the hand. The second or negative phase of the tactile functional test consists in occluding each of the patient's nostrils alternately with the ball of the thumb and instructing him to breathe deeply and vigorously through the open nostril. By this method it will be found that when there is a marked nasal obstruction on one side, while the other nostril functions normally, the examiner will experience a distinct pull on the occluding thumb.

Evaluation and Interpretation of the Two Phases of the Test:

The first or positive phase of this procedure is entirely misleading. In the first place, it renders information only on the expiratory aspect of breathing, whereas our chief concern is the problem to ascertain the permeability of the nasal passages insofar as it involves the sufficiency of the air intake during the act of inspiration. Were we to judge about the patient's expiratory effort from the force with which the outgoing air current strikes our hand, we would be greatly deceived. In drawing comparisons based on the force of the air current striking the hand, from a partially obstructed nostril and from a functionally normal nostril, it will often be found that the greater impact is experienced on the side of the obstruction. The reason for this seemingly contradictory phenomenon may be explained on the basis that the force of the air current emanating from the defective nostril is concentrated or focused on a smaller area of the receptive surface, whereas the air coming from the normal side is diffused over a wider area of the receptive surface and, therefore, is felt less intensely. Another reason for this paradox may lie in the fact that when a patient is told to exhale through an obstructed or partially occluded nostril, unintentionally he will exert a greater effort in the expulsion of the air through a partially obstructed nostril than through a nor-

mally functioning one and, therefore, the air current from the defective nostril will strike the hand with greater force than that coming from the normal side.

The negative phase of this test, on the other hand, is of distinct value in the appraisal of nasal obstructions. Especially is this true when one nostril is greatly defective and the opposite one is normal or near normal. When the examiner's finger is made to close the normal nostril and the patient is instructed to breathe vigorously through the other one, the examiner will experience a distinct upward pull on the occluding surface of his finger. If, contrariwise, the defective nostril is closed with the finger and the patient breathes through the normal nostril no such sensation will be experienced.

(c) Visual: In watching the alae nasi during the act of respiration it will be observed that they move under certain conditions. In people with very delicate and sensitive nostrils the alae nasi will be seen to move slightly with every respiratory act. In cases of labored breathing the alae will move quite perceptibly even in persons with thick and insensitive nostrils. These movements furnish definite and valuable information on the status of the nasal respiratory function. The alar excursions may vary from a barely perceptible motion in cases of normal breathing, or where there is a slight obstruction to complete collapse of nostril so that it comes to rest on the nasal septum in cases where the occlusion markedly interferes with nasal respiration. The procedure is as follows: The examiner sits opposite the patient and a reflected light is directed on his nose while the examiner observes the movements of the alae nasi during quiet and vigorous breathing, first through both nostrils and then through each nostril separately, by closing each of the patient's nostril's alternately and comparing the movements of the alae with one another. It will be observed in cases of unilateral obstructions that when the defective nostril is occluded with the finger and the patient breathes through normal side there will be a very slight or barely perceptible movement of the ala on that side, not on the side of the obstruction. When, on the other hand, the normal nostril is closed with the finger and the patient is made to breathe through the defective nostril there will be a distinct caving in of both nostrils, more so on the normal side.

Interpretation and Evaluation of the Visual and Tactile Phenomena: During the course of normal nasal breathing, pressure or tension is generated within the nasal chambers. With every expiration a positive pressure is engendered within the nasal passages, and with every expiratory act negative pressure is set up in the same. This pressure or tension exerts its influence upon the alae nasi, causing them to move with each respiratory act. The degree of intranasal pressure and the excursions of the alae nasi which are influenced by it depend upon several factors: the vigor of the respiratory act, the patency of the nasal air passages, and to these may be added the sensitivity of the nostrils in determining the movements of the ala. The greater the force of respiration the greater will be the pressure within the nose. Again, when a nostril is partly or completely blocked by some obstruction, a partial or almost complete vacuum is created within the nose (on the side of the obstruction) so that when he (the patient) is made to breathe through it (the opposite side being closed by the examiner's finger), the alae nasi will collapse and come to rest on the nasal septum.

While the tactile and visual data furnished by close observation are of great importance in the functional examination of the nose (and they furnish the sum total of our functional tests), yet they fail to establish a norm or standard for normal or abnormal nasal respiratory function and as such there is much to be desired in this respect.

(d) Narometry: This is best defined by stating that it is a method of determining nasal permeability by measuring (by means of a narometer* — presently to be described — the amount of negative pressure created within the nasal chambers during the inspiratory phase of respiration, or the method of translating the visible and tactile data of the rhinologic functional tests into a measurable quantity — millimetres of mercury.

Description of Instrument: This consists of a U-shaped glass tube mounted on a stand against a white-surfaced background of bakelite. The left branch of the tube without any markings extends for about four inches to an open end, which is covered by a screw cap for the purpose of cleaning and the

*Made by Becton, Dickinson and Co., Rutherford, N. J.

entrance of mercury. The right limb of the tube, beginning about an inch and a half from the bend, is calibrated in 2 mm. markings, starting at zero and extending up the tube to 60 mm. The calibrations on the tube are marked with numerals at each 10 mm., beginning with zero and proceeding with 10, 20, 30, 40, 50 and 60. The right or calibrated limb of the tube extends to the top, where it is turned at right angles, ending in a nipple-like projection of about an inch in length. Over this projection a soft rubber tube of about 30 to 36 inches is fitted, and in the distal end of the tube a hard rubber nasal tip is inserted.

Method of Procedure in the Use of the Narometer: The patient is seated opposite the examiner and the narometer is placed on a tray, table or desk with the calibrated surface in full view of the examiner. The patient is instructed to clear his nose of any accumulation of mucus or pus that may be present at the moment in his nasal chambers. The nasal tip of the narometer is now inserted gently but snugly into either nostril of the patient so as to completely shut off its opening, making sure that no air can seep through alongside the nasal tip, in which event the test would become vitiated. Care must likewise be exercised when inserting the nasal tip not to press it too hard, and making sure that its opening does not impinge against any part within the nostril, thus interfering with the free circulation of the air current, which would likewise render the test inaccurate. Now, the patient is told to breathe normally and then forcefully through the open nostril with his lips tightly closed while the examiner watches the head of the mercury column in the calibrated limb of the narometer. The examiner should bear in mind that during this procedure it is the free nostril that is being tested.

In normal bilateral nasal respiration it will be observed that when the nasal tip of the narometer is inserted into either nostril and the patient breathes deeply and evenly through the open nostril there will be an almost imperceptible vacillation of the mercury column, ranging from a fraction of a millimetre to 1 mm. If the patient has one normally functioning nostril and one defective nostril, on inserting the nasal tip in the defective nostril, with the patient breathing through the normal nostril the head of the mercury col-

umn will rise slightly from zero to about 1 mm. If, on the contrary, the nasal tip of the narometer is inserted into the normal side and the patient breathes through the defective nostril there will be a sharp rise in the mercury column, ranging 2 mm. to 10, 20 or even 60 mm., depending upon the degree of obstruction and upon the vigor of the patient's general respiratory capacity. If both nostrils are defective the narometer will reveal it by a mercury rise above the normal when the tip is introduced in either of them and the usual procedure is followed. In bilateral normal nasal function, if the nasal tip of the narometer is introduced in either nostril and the opposite nostril is occluded by the examiner's finger and the patient is instructed to breathe deeply through his mouth, the mercury column will register a slight rise, between a fraction to 1 mm., the same as in breathing through a normal nostril. This proves that normal nasal respiration through each nostril equals that of oral respiration.[1]

Now, the question posed as the introduction of this paper, "What Constitutes Normal Nasal Respiration?" can be answered in terms of millimetres of mercury. Any rise in the column of mercury up to 1 mm. under the conditions outlined constitutes normal nasal respiration. A rise of the mercury column between 1 and 2 mm. constitutes a borderline case. Any rise above this definitely denotes a deficiency in nasal breathing.

REFERENCE.

1. BECKER, B. M.: The Respiratory Function of the Nose and Nasal Obstructions. THE LARYNGOSCOPE, 42:695, Sept., 1932.

2000 84th Street.

A REVIEW OF THE ARTICLES ON TUBERCULOSIS IN THE FIELD OF OTOLARYNGOLOGY FOR LATE 1941 AND EARLY 1942.

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During 1942 there has been the greatest dearth of articles on tuberculosis since the war began in August, 1939, and for many years before that. This is due to our own entrance into the war, as well as our inability to get foreign medical journals. A few articles have appeared in foreign journals which have not been received in the large medical libraries of this country; however, very few articles have been published even if all the journals were available. A few years ago, 72 articles were available for review and last year, 42. This year only 12 are available.

In time of war, clinical investigation may and does suffer in some respects, although great progress is often made in war surgery with improved methods of treatment for war wounds. Research suffers more, and this is inevitable because we must and will win the war.

This year the articles have almost nothing very new, although a few minor advances have been made. In the past the literature has usually contained two or three excellent articles which have advanced our knowledge of diagnosis and treatment.

Brahy[1] summarized his experiences in the treatment of laryngeal tuberculosis over a period of 13 years, during which he was able to follow thousands of cases.

Silence on the part of the patient is the essential prerequisite of any treatment according to this author. Unfortunately, most patients fail to grasp the importance of this measure. It is good practice, in order to guard against laryngeal involvement in pulmonary tuberculosis, to enjoin every patient affected with the latter to refrain from talking. Medicated throat washes, not necessarily of high concentration, used several times a day, rid the larynx of the mucopurulent bron-

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chial secretions, and so remove the danger of contact infection. Application, in one form or another, of various oils (gomenol, menthol, cod liver oil, chaulmoogra oil, etc.), lactic and trichloracetic acids, failed in the hands of the author; moreover, he found every one of these procedures very trying for the patient and often the cause of severe coughing or even glottic spasm. Orthoform and cocaine gave satisfactory symptomatic relief. At the stage of odynophagia, control of pain, lasting from several hours to several weeks, was obtained by applications of 5 per cent cocaine (vaporized) to the throat and injection of novocain and alcohol into the superior laryngeal nerve in the interhyothyroid space. The treatment may be repeated as needed.

Light therapy, both natural and artificial, gave good results, but is difficult to apply, either because of the complexity of the apparatus or owing to lack of co-operation on the part of the patient.

Resection of the superior laryngeal nerve also gave relief in the terminal stage; in contrast to novocaine and alcohol injections, the result is permanent. Tracheotomy assures resting of the larynx but few patients consent to such an operation. Electrocoagulation was effective in diffuse edema of the arytenoid muscles. The galvanocautery was the procedure of choice in ulceration and interarytenoid proliferation, where it often produced amazing results. The treatment was conditioned on absence of fever, unilateral involvement of the lungs, and on a good fibrous reaction on the part of the tissues. There are no general indications, as each and every case must be considered individually.

The chances of cure or, in any event, amelioration are relatively slight in tuberculosis of the larynx, but the condition is by no means hopeless. The extent of pulmonary involvement is the deciding factor. Phrenicectomy, pneumothorax and other surgical procedures in the chest are valuable adjuvants. Chédé's operation, thoracoplasty and lobectomy prevent the disease from reaching the stage of laryngeal involvement or in any event enable us to treat these rare cases in a satisfactory fashion.

Raimondi and Nijensohn[2] reported a case of Isambert's disease; *i.e.*, miliary tuberculosis with predominant oral, pharyn-

geal and laryngeal localization. This was presented as an addition to other cases previously reported in Argentina. The family history of the patient, a male, age 34 years, presented tuberculosis; seven years previously, the patient had suppurative cervical adenitis, which was cured by radiotherapy.

The onset of the reported condition, three months earlier, was marked by pain on swallowing; later, pain became spontaneous, and the patient lost weight and labored under nocturnal coughing. Biopsy of the left tonsil showed follicular tuberculous lesions. There was no history of physical or emotional trauma.

Ilyish[3] presented the cases of over 80 patients with extensive tuberculous affections of the pharynx, larynx, tongue, cheeks, lips, epiglottis, etc. He applied freezing in all these cases for short periods in the beginning of 10-30 seconds, gradually increasing the time to three, five or seven minutes. In the regions of the hard palate, nasal septum and gums, the time was considerably shorter. The interval between the first and second treatment was from 10 to 20 days.

The freezing effected good results in all cases with the exception of those where the general state was unfavorable from the beginning. The above method is especially recommended in tuberculous affections of the vocal cords and slight infiltrations of the pseudovocal cords, with surface ulceration, in circumscribed infiltrations of the arytenoid cartilages and interarytenoid space.

Contraindication for application of freezing is only the total absence of immunobiological properties of the organism with symptoms of negative allergy; nevertheless, in grave cases accompanied by dysphagia, careful dosage of freezing may effect alleviation of pain. In acute outbreaks of hematogenous processes in the larynx with benign course (in most cases with simultaneous outbreak of the pulmonary process) carbon dioxide snow must not be applied until after the natural retrogression of the inflammatory symptoms; intervention is indicated only by the presence of residual manifestations on the circumscribed area or in cases of marked dysphagia. The technique is simple, although it requires a certain knowledge, as in all general endolaryngeal manipulations, and should be obligatory to all phthisiolaryngologists according to the author.

Fedorov's[4] paper covered 16 cases of Roentgen treatment of tuberculosis of the larynx. The author used combined irradiation; in the begnning the Roentgen rays were applied to the sympathetic cervical ganglia and later, local irradiation was applied. The average doses were from 6-8 or 12-15 per cent HED.

According to the nature of the tuberculous process the cases were classified as follows: fibro-productive compensated pulmonary tuberculosis, four cases; fibro-cavernous, slowly progressing, eight; and productive, exudative decompensated, four. The nature of the laryngeal processes was in 12 patients, initial infiltrative ulcerous affections, predominantly of a productive nature, and in four patients, exudative processes in the phase of infiltration and degeneration. Therapeutic results (convalescence): *1.* Marked improvement, two; general improvement, six; and without changes, seven. All cases which showed improvement belonged to the group with a predominant productive nature of the affection with inhibitory tendencies. Out of nine patients who showed improvement, two died a year later due to fulminating pulmonary laryngeal processes; five patients were under observation for periods from three to six months to one year, the process remaining in a certain stabilized phase. The above results confirm the antidysphagic action of Roentgen rays. In 12 patients with dysphagia in some degree, the latter was reduced following irradiation; nevertheless, complete reduction cannot be achieved by one application of the rays. Out of nine patients who improved, five showed contraindications to the galvanocautery and other methods of active therapy.

Although the author's material was limited, it allows us to consider Roentgen therapy as having an important place among other methods in the treatment of tuberculosis of the larynx.

According to Fadeeva[5] the most responsive patients to Roentgen therapy were those with reduced and chronic, slowly progressing forms of hematogenous disseminated laryngopulmonary tuberculosis without symptoms of general intoxication. In the larynx of such patients there was generally a predominance of productive and proliferative elements; exudations and caseous necrosis were circumscribed.

Roentgen therapy was chosen in forms that were generally followed or threatened by stenosis.

Patients less suitable for Roentgen therapy were those with chronic but markedly progressing processes with unstable general compensation and even slight but stable subcompensation. In the majority of these patients, Roentgen therapy, together with the climatic and sanatorium conditions of the southern shore of the Crimea, was able to check and reduce the progression of the process, but it was seldom that it produced a stable regression.

Contraindications: All patients with subacute progressing forms of hematogenous disseminated laryngopulmonary tuberculosis, and chronically progressive with symptoms of marked general intoxication.

Roentgen therapy demanded an accurate classification of the patients in the selection of the site and dosage of irradiation.

Hall, Pfuetze, Hinshaw and Feldman[6] have reported the effect of promin on the blood of tuberculous patients. They found that the drug inhibits the development of experimental tuberculosis in guinea pigs. They also found it necessary to give the drug over a long period of time to avoid the anemia, which was apparently hemolytic in type, when promin was given in large doses. The authors have listed many chemicals which have this hemolytic action and especially aniline derivatives such as promin. The blood must be carefully watched when promin is given.

Seventy patients are included in their study. Sixty-four of the 70 had various types of pulmonary tuberculosis. In most cases the drug was given by mouth. A few patients were treated parenterally. The action of the drug was more pronounced when given by mouth. Doses of 1.6 to 3.2 gm. daily by mouth were tolerated for eight to 10 days by most patients. After 10 days the hemoglobin and erythrocyte count fell slowly or rapidly. The rapidity with which the blood returned to pretreatment levels, after treatment was stopped, was often remarkable and often occurred in a few days.

Intermittent treatment was safer than continuous treatment as the blood-forming organs were able between treatments to re-establish the pretreatment blood levels of hemo-

globin and red cells. The authors also found that starting with small doses and gradually increasing, there was developed an increased tolerance for the drug, presumably due to the hyperplasia of the bone marrow; this avoided severe degrees of anemia.

Adams[7] found tuberculosis of the middle ear insidious and often not recognized until late in the disease. He called attention to the importance of the different forms of collapse therapy for the treatment of pulmonary tuberculosis and as a valuable means of preventing the aural complication; however, he found the complication of otitis media more frequent since 1938. He repeatedly examined two patients over a period of many months. One patient had a hearing loss of 30 per cent and the other, 40 per cent. He described the symptoms as these developed from week to week until discharge appeared in the external auditory canal. He compared the objective symptoms to those of an acute catarrhal otitis media. Most of the patients had a sense of fullness with mild tinnitus. He mentioned infection of the middle ear by way of the Eustachian tube and also through the blood stream. All of the 10 patients observed by the author had patent Eustachian tubes. He believed this favored tuberculous otitis media.

Searcy, McBurney and Rowe[8] believed a relationship exists between the acid-fast tubercle bacillus and the ray-fungus. One of the authors (Searcy) has treated actinomycosis of the jaw successfully with thymol by mouth. When thymol was not well tolerated by the stomach, he gave intramuscular injections of the drug in olive oil. The authors mentioned the toxicity of thymol, but they used it for tuberculous patients. Each patient took one 5 gr. capsule of thymol after each meal with a glass of whole milk to facilitate absorption. There was no change of environment or of the dietary regimen. At the time of reporting the results the patients had been under treatment for nine weeks. Not one of the patients had experienced any ill effects from the thymol. They had six case reports in their article. The authors realized they had too few cases and not a long enough period of time for observation before making their report. All of the patients were improved by the treatment with thymol.

Proctor and Lindsay[9] have reviewed briefly the early au-

thors' articles on tuberculosis of the middle ear. They mention the incidence of tuberculous otitis media in different clinics. The authors stressed the histopathology of the disease. They reported in detail eight cases and they showed 13 microphotographs. The article is rather long and very complete. It should be read in its entirety, as it is too long to abstract and do justice to the article.

The authors mentioned the complications of the disease, such as single or multiple fistulas, facial paresis or paralysis, labyrinthitis, tuberculous pachymeningitis, erosion of the internal carotid artery or jugular bulb, osteomyelitis and extension to the temporomandibular joint. Diagnosis and treatment were only briefly discussed. The major portion of the article dealt with histopathology.

Ellwood, Potter, Sacco and Pagliughi[10] believed the control of pulmonary tuberculosis was important when tracheobronchitis coexisted. They referred to the numerous articles in the literature during the past five years dealing with tuberculous tracheobronchitis and the better understanding of this complication of pulmonary tuberculosis. They found some controversy in the literature concerning the best methods of treatment, such as collapse therapy or local measures.

The report was based on 302 bronchoscopic examinations of 137 selected patients with pulmonary tuberculosis. Th authors found tracheobronchitis in 79 patients, or 58 per cent. There were seven tables and nine references to the literature.

The authors found the patients improved with local treatment, but that cicatricial stenosis could not always be prevented. A few patients recovered without treatment. All of the work was carried out by clinicians and bronchoscopists. In conclusion the authors emphasized the importance of controlling the pulmonary lesions.

The abstract of Mandadori and Faria Pereira[11] as it appeared in the *Journal of the American Medical Association* was "Electroanalgesia in Tuberculous Painful Dysphagia." The abstract was as follows: "Mandadori and Faria Pareira[11] direct attention to Grain's method of 'electroanesthesia (iodine ionization) of the larynx' and the value of the procedure in

the therapy of acute painful swelling in tuberculous laryngitis. The metallic electrodes of steel and tin, respectively, are bent to the form of the throat and back of the neck. The concave part of each electrode is filled with a thick layer of cotton. The anterior electrode is soaked in a 2 per cent potassium iodide solution and the posterior one in water. The electrodes are firmly applied to the throat and back of the neck by means of an elastic bandage. A galvanic current of 12 milliamperes is passed through the electrodes for 30 minutes. Pain stops completely within the first 15 minutes of the treatment. It reappears within or after 24 hours. In either case the treatment is repeated only when pain recurs. The intervals of recurrence of pain after the first 24 hours rapidly increase from several days to several weeks. All patients can resume normal feeding immediately after the first treatment. The treatment controls pain permanently without any effect on the course of the tuberculous laryngeal lesions. Edema of the larynx is the only contraindication. Administration of local and inhalation anesthesia and local trauma of any degree are prohibited, as they nullify the effects of electroanalgesia. The treatment is painless, harmless and simple. It gives good results in all cases, regardless of the stage and extension of the involvement." It was impossible for the reviewer to obtain the original article.

Mann[12] emphasized the prevalence of pulmonary tuberculosis and that likely one out of every three has some laryngeal disease. He mentioned the prevalence of the laryngeal complication from 20 to 40 years of age. The modes of infection of the larynx by sputum, through the lymphatics or blood stream, were discussed. The infection usually manifests itself intrinsically at the interarytenoid region or on the vocal cords. The arytenoids, aryepiglottic folds and epiglottis constitute the extrinsic parts of the larynx.

The symptoms such as huskiness, dryness, alteration of the voice, dysphagia, dyspnea, etc., were emphasized by the author and given due importance.

The prognosis he found dependent upon the laryngeal disease in extent and location, the extent and location of the pulmonary lesion and the general health of the patient.

Under treatment, the general, pulmonary and laryngeal

received due consideration. He emphasized rest of the voice, ultraviolet therapy and the cautery. The article had 24 references.

BIBLIOGRAPHY.

1. Brahy, J.: Traitement actuel de la tuberculose laryngée. *Union méd. du Canada*, 70:954-955, 1941.

2. Raimondi, A. A., and Nijensohn, M.: Presentación de un caso de tuberculosis miliar faringolaringéa. *Arch. argent. de fisiol.*, 17:336-339, 1941.

3. Ilyish, A. R.: Treatment of Laryngeal Tuberculosis with Carbon Dioxide Snow. *Sovet. med., Moskva*, 5:13-14, 24, 1941.

4. Fedorov, N. D.: Results of Roentgentherapy of Laryngeal Tuberculosis. *Probl. tuberk.*, 7-8:63, 1941.

5. Fedorov, A. K.: Roentgenotherapy of Hematogenous Disseminated Laryngo-Pulmonary Tuberculosis at the Southern Shore of the Crimea. *Probl. tuberk.*, 6:77-82, 1941.

6. Hall, B. E.; Pfuetze, Karl; Hinshaw, H. C., and Feldman, W. H.: Effect of Promin on the Blood of Patients with Tuberculosis. *Proc. Staff Meet., Mayo Clinic*, 17:24-27, Jan. 14, 1942.

7. Adams, John G.: Tuberculous Otitis Media: A Complication of Thoracoplasty. *Ann. Otol., Rhinol. and Laryngol.*, 51:209-214, March, 1942.

8. Searcy, Harvey B.; McBurney, Ralph, and Rowe, Harry S.: Thymol Therapy in Tuberculosis. *Jour. Med. Assn. of Alabama*, Jan., 1942.

9. Proctor, Bruce, and Lindsay, J. R.: Tuberculosis of the Ear. *Arch. Otolaryngol.*, 35:221-249, Feb., 1942.

10. Elwood, B. J.; Potter, B. P.; Sacco, A. G., and Pagliughi, J. J.: Importance of Control of a Pulmonary Lesion When Tuberculous Tracheobronchitis Coexists. *Arch. Otolaryngol.*, 35:408-417, March, 1942.

11. Mandadori, E. C. F., and Faria Pereira, F.: Traumatic Symptomatic Painful Dysphagia Treated by Electric Analgesia. *Jour. A. M. A.*, p. 377, May 23, 1942.

12. Mann, Bertram T.: Laryngeal Tuberculosis. *Practitioner*, 148:231-235, April, 1942.

Physicians' Building.

BOOK REVIEW.

Ear, Nose and Throat Nursing. By Jas. Hardie Neil, D.S.O., C. de G., F.R.C.S., F.A.C.S., Ear, Nose and Throat Surgeon, Auckland Hospital and Mater Misericordiae Hospital, Auckland; Past President, Pan-Pacific Surgical Congress. Third Edition. One hundred twenty-seven pages with index and numerous illustrations.

This is a short paper-covered book written primarily for the instruction of nurses in ear, nose and throat techniques. It is well illustrated and practical points in the preparation of instruments for operation and the application of dressings, etc., are emphasized.

This book is a simple and clearly written handbook on diseases of the ear, nose and throat with special attention to practical points in nursing technique. It is recommended to all student-nurses and might be used by instructors as a guide to lectures to such nurses. T. E. W.

Nasal Medication. A Practical Guide. By Noah N. Fabricant, M.D., M.S., Associate in Laryngology, Rhinology and Otology, University of Illinois College of Medicine. One hundred twenty-two pages with index and numerous illustrations. Baltimore: Williams and Wilkins Co. 1942. Price $2.50.

Dr. Fabricant has prepared a particularly useful book to guide rhinologists and general practitioners in the use of nasal medications. In the first four chapters he has supplied a helpful and concise guide to the anatomy, histology and physiology of the nose and sinuses; he discusses the effects of drugs on the mucous membrane and also their effects on other parts of the body. He also discusses topical applications in acute and chronic diseases and has given a full bibliography on the subject.

The author's work on the effect of the P_H of solutions on nasal function is well known and Chapter 5 of this book is of particular interest.

In the preface Dr. Fabricant states that the purpose of the book is to serve as a practical guide to physicians and to provide a quick and ready reference for those who employ nasal medication. He has indeed fulfilled his objective, and this volume is recommended not only to rhinologists but also to students and general practitioners as well. T. E. W.

Diseases and Injuries of the Larynx, A Textbook for Students and Practitioners. By Chevalier Jackson, M. D., Sc.D., LL.D., F.A.C.S., Honorary Professor of Broncho-Esophagology, Temple University, Philadelphia, and Chevalier L. Jackson, A.B., M.D., M.Sc. (Med.), F.A.C.S., Professor of Broncho-Esophagology, Temple University, Philadelphia. With over 200 illustrations and 11 plates in color. New York: The MacMillan Co. 1942.

The authors have compiled a volume which covers the subject of laryngeal injuries and diseases completely and fully. The book is profusely and excellently illustrated, the color plates in particular being well reproduced and of great value.

The chapters on acute and chronic stenosis of the larynx and acute and inflammatory diseases of the larynx are recommended not only to laryngologists but to the general practitioner and pediatrician as well. The chapter on war surgery of the larynx is particularly timely.

This book is indeed a textbook on diseases and injuries of the larynx and is strongly recommended to all postgraduate students and laryngologists and as a reference book in every medical library. T. E. W.

CASSELBERRY AWARD OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION.

A sum of money is now available from the Casselberry Fund of the American Laryngological Association for a prize award, decoration or grant to be given for original investigation in the art and science of Laryngology or Rhinology. Theses must be in the hands of the Secretary, Dr. Arthur W. Proetz, 1010 Beaumont Medical Building, St. Louis, Mo., before March 1, 1943.

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